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OUTCOME IN NEONATES BORN TO MOTHERS WITH RUPTURE OF MEMBRANES OF VARYING DURATION

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Dr. SHUBHANGI BANSAL

ABBREVIATIONS

Apgar	-	Appearance, Pulse, Grimace, Activity, Respiration
BMI	-	Body Mass Index
CFR	-	Case Fatality Rate
CRP	-	C-Reactive Protein
EONS	-	Early Onset Neonatal Sepsis
G1	-	Gravida 1 (First Pregnancy)
G2	-	Gravida 2 (Second Pregnancy)
Hb	-	Hemoglobin
HMD	-	Hyaline Membrane Disease
IUGR	-	Intrauterine Growth Restriction
IVH	-	Intraventricular Hemorrhage
LRTI	-	Lower Respiratory Tract Infection
MAS	-	Meconium Aspiration Syndrome
MCH	-	Mean Corpuscular Hemoglobin
MCHC	-	Mean Corpuscular Hemoglobin Concentration
MCV	-	Mean Corpuscular Volume
MS Excel	-	Microsoft Excel
NEC	-	Necrotizing Enterocolitis
NICU	-	Neonatal Intensive Care Unit
NPV	-	Negative Predictive Value

OPD	-	Outpatient Department
POG	-	Period of Gestation
PPROM	-	Preterm Premature Rupture of Membranes
PROM	-	Premature Rupture of Membranes
PVL	-	Periventricular Leukomalacia
RDS	-	Respiratory Distress Syndrome
ROM	-	Rupture of Membranes
SD	-	Standard Deviation
TLC	-	Total Leukocyte Count
TTN	-	Transient Tachypnea of the Newborn

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INTRODUCTION

INTRODUCTION

Premature rupture of membranes (PROM) is a condition where the amniotic sac ruptures before the onset of labor, compromising the protective environment surrounding the fetus. It is categorized by gestational age into term PROM, occurring at or after 37 weeks of gestation, and preterm PROM (PPROM), which occurs before 37 weeks. These classifications are critical as they guide both the management strategies and the potential outcomes associated with the condition [1].

Incidence and Epidemiology

Premature rupture of membranes (PROM) is a frequent complication during pregnancy, occurring in approximately 8-10% of pregnancies at term [2]. A more severe form, preterm PROM (PPROM), affects 2-4% of all pregnancies and is a major cause of spontaneous preterm births, accounting for nearly 40% of these cases [3]. Both conditions pose significant risks to maternal and neonatal health, contributing to around 10% of perinatal deaths globally.

The incidence of PROM is influenced by geographical and socioeconomic factors. In high-income countries, access to advanced healthcare systems generally leads to better outcomes. In contrast, resource-limited settings face heightened risks of complications due to limited medical infrastructure and resources. Prematurity remains a pressing global health challenge, with millions of neonatal deaths occurring each year, many of which are linked to preterm birth and its associated complications [4].

Factors

Various factors can contribute to the occurrence of premature rupture of membranes (PROM). Infections, particularly those affecting the genital tract, such as bacterial vaginosis and sexually transmitted infections, can weaken the membranes, making them more susceptible to rupture. Certain medical conditions, including cervical insufficiency, a history of PROM in previous pregnancies, and multiple gestations, are also associated with an increased risk [5].

Lifestyle choices and overall health play a significant role as well. Smoking, inadequate nutrition, and a low body mass index (BMI) are notable contributors to the likelihood of PROM. Additionally, physical trauma or invasive medical procedures, such as amniocentesis, can lead to premature rupture [6]. In some instances, however, the exact cause remains unknown, complicating efforts to develop effective prevention strategies.

Impact

The effects of premature rupture of membranes (PROM) depend largely on the gestational age at which it occurs and the duration of the rupture. For mothers, prolonged PROM lasting more than 18 hours significantly increases the likelihood of infections such as chorioamnionitis, posing serious health risks [7].

For neonates, the consequences can be even more severe, particularly in premature births. Complications include respiratory distress syndrome (RDS), brain hemorrhages, sepsis, and long-term developmental issues [8]. A prolonged rupture also heightens the risk of neonatal infections, including sepsis, which can further compromise outcomes.

PROM triggers an inflammatory response that can accelerate fetal lung maturation, potentially benefiting the infant. However, excessive inflammation can damage lung tissue and increase the risk of conditions such as bronchopulmonary dysplasia. Additionally, preterm births resulting from PROM are a leading cause of neonatal morbidity and mortality, especially in cases where delivery occurs before 32 weeks of gestation [9].

Management

The management of premature rupture of membranes (PROM) involves carefully weighing the risks of prematurity against those associated with prolonged rupture. In cases of preterm PROM (PPROM) before 34 weeks of gestation, expectant management is often employed. This approach includes hospitalization, close monitoring, and interventions aimed at prolonging the pregnancy while minimizing the risk of infection [10]. Administering antibiotics is a key strategy to reduce the

likelihood of ascending infections, while corticosteroids are used to promote fetal lung development, enhancing the chances of neonatal survival. Magnesium sulfate may also be given to protect the fetal brain and reduce the risk of neurological complications.

For term PROM or when complications arise, active management is typically recommended. This may involve inducing labor or performing a cesarean delivery to minimize risks to both the mother and the baby. Postnatal care is equally important, focusing on screening for neonatal infections, providing respiratory support if needed, and ensuring long-term developmental follow-up, particularly for premature infants, PROM and PPROM pose significant challenges during pregnancy due to their complex maternal and neonatal risks. Timely recognition, appropriate medical management, and thorough postnatal care are essential to improving outcomes and reducing complications for both the mother and the newborn [11].

The aim of this study is to evaluate the outcomes in neonates born to mothers who experienced premature rupture of membranes (PROM) for different durations: 18-24 hours, 24-72 hours, and more than 72 hours. The primary objective is to assess neonatal outcomes based on these timeframes. The secondary objectives include identifying the association between maternal risk factors and neonatal outcomes following PROM, examining the impact of the latency period between PROM and delivery on neonatal outcomes, and determining the case fatality rate among neonates born to mothers with PROM lasting more than 72 hours.

AIMS AND OBJECTIVES

AIM AND OBJECTIVES

Aim:

To evaluate outcome in neonates born to mothers with premature rupture of membrane of 18-24hrs, 24-72hrs and >72hrs.

Objectives:

Primary Objective:

To evaluate outcome in neonates born to mothers with premature rupture of membrane of 18-24hrs, 24-72hrs and >72hrs.

Secondary Objective:

- To find out association between maternal risk factors and neonatal outcome following PROM.
- To study the latency period between PROM and delivery on neonatal outcome.
- To find out the case fatality rate among the neonates born to mother with PROM of > 72 hrs duration.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Premature Rupture of Membranes (PROM) refers to the early rupture of the amniotic sac, which normally holds the fetus and amniotic fluid. In most cases, the sac breaks during labor when contractions are already underway, but in PROM, it ruptures before labor begins. This premature rupture can happen at any point during pregnancy and poses certain risks to both the mother and the baby. When the amniotic sac ruptures, the protective barrier is lost, potentially increasing the risk of infections like chorioamnionitis. PROM requires prompt medical attention to determine the appropriate management strategy, which may involve waiting for labour to start naturally or inducing labour if necessary to reduce infection risks [12].

A specific form of PROM, known as Preterm Premature Rupture of Membranes (PPROM), occurs when the membranes rupture before 37 weeks of gestation, putting the fetus at higher risk of complications associated with premature birth. PPROM is one of the leading causes of preterm births, and the earlier it occurs, the more serious the potential consequences. Babies born prematurely may experience a range of complications, including underdeveloped organs, particularly the lungs, which can lead to breathing difficulties [13]. In addition to preterm birth, PPROM can increase the chances of infection, umbilical cord prolapses (where the cord slips into the birth canal), and pulmonary hypoplasia, a condition in which the foetus's lungs do not develop properly.

The management of PROM and PPROM depends on the gestational age and the health of both mother and baby. When PROM occurs after 37 weeks of gestation, if labour does not begin on its own shortly after the rupture, doctors may recommend inducing labour to prevent infections. However, when PPROM occurs before 37 weeks, the priority is often to prolong the pregnancy safely while minimizing the risks. This may involve hospital admission, administering antibiotics to prevent infection, and giving corticosteroids to help the foetus's lungs mature in case of an early delivery. Throughout the process, the health of both mother and baby is carefully monitored to determine the best course of action for a safe outcome [14].

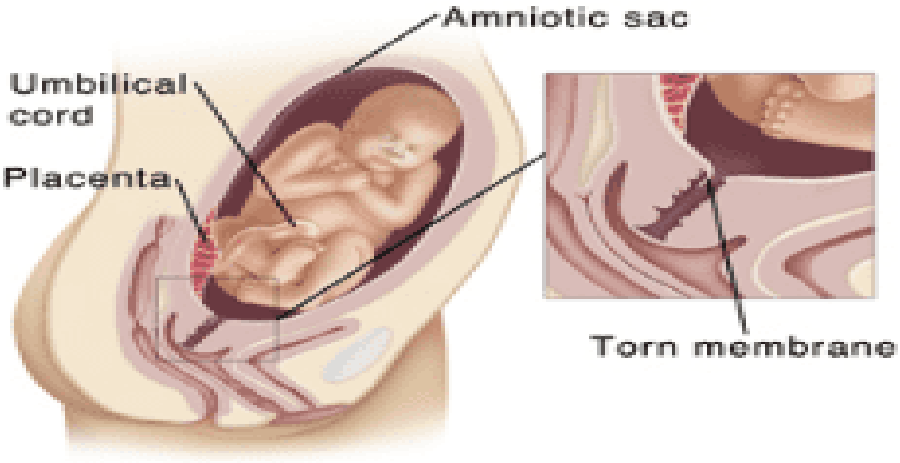

Figure 1: Premature Rupture of Membranes

(https://www.medical-actu.com/en/premature-rupture-of-membranes/)

Risk Factors Associated with PROM

The occurrence of premature rupture of membranes (PROM) is influenced by several factors, some related to the mother's health and others to the pregnancy itself. One significant contributor to PROM is maternal infection. Conditions like bacterial vaginosis, chlamydia, and urinary tract infections can weaken the amniotic sac, making it more susceptible to rupture. These infections may lead to chronic inflammation, which compromises the integrity of the fetal membranes. The inflammatory process triggered by infections can produce enzymes that degrade the structural components of the membranes, such as collagen, ultimately weakening them over time and increasing the risk of rupture [15].

A woman's previous obstetric history also plays a role in the risk of PROM. Women who have experienced PROM or preterm birth in past pregnancies are at higher risk of facing similar complications in future pregnancies. This could be due to underlying anatomical or physiological conditions, such as cervical or uterine abnormalities, which make the membranes more vulnerable to rupture. Additionally, certain unresolved infectious or inflammatory processes from earlier pregnancies may continue to contribute to the risk. In some cases, there may also be genetic predispositions to membrane weakness, making these women more prone to PROM [16].

Table 1: PROM Influencing Factors

Contributing Factors	**Description**
Maternal Infection	Infections weaken membranes through inflammation and enzyme activity.
Previous Obstetric History	History of PROM or preterm birth increases future risk due to physiological or anatomical factors.
Lifestyle Factors	Smoking and substance use reduce membrane strength and increase infection risk.
Maternal Age	Young and older mothers have higher risk due to hormonal imbalances and collagen degradation.
Cervical Conditions	Short cervix or past surgeries weaken membranes, increasing rupture risk.
Pregnancy-related Factors	Polyhydramnios and multiple pregnancies increase pressure on membranes.
Medical Procedures	Procedures like amniocentesis or cerclage can irritate or puncture membranes.
Trauma to the Abdomen	Physical trauma can directly damage membranes or increase uterine pressure.

Lifestyle factors, such as smoking and substance use, further elevate the risk of PROM. Smoking during pregnancy weakens the immune system, increasing susceptibility to infections that could compromise the membranes. Smoking also reduces blood flow to the placenta, which can affect the health and integrity of the fetal membranes. Illicit substances like cocaine have also been shown to weaken placental attachment and reduce membrane strength, creating a higher likelihood of rupture. These substances can disrupt the normal hormonal balance and biochemical environment required to maintain membrane strength throughout pregnancy.

The age of the mother is another factor that influences the risk of PROM. Younger mothers, particularly adolescents, and women of advanced maternal age are more vulnerable [17]. In adolescents, ongoing physical development and hormonal fluctuations may affect the strength of the membranes. For older women, age-related

changes in collagen and other proteins in the body can make the membranes less elastic and more prone to tearing under the pressure of a growing fetus. As a result, both age groups may experience a higher rate of PROM due to these physiological factors.

Cervical conditions, particularly a short cervix or cervical insufficiency, are also key contributors to the risk of PROM. When the cervix is too short or prematurely opens, it places increased pressure on the fetal membranes, making them more likely to rupture. In women who have undergone procedures like conization, in which part of the cervix is removed, the risk of PROM is heightened because these surgeries can weaken the cervix's ability to support the pregnancy. The combination of cervical weakness and the increased pressure on the membranes can significantly raise the chances of rupture.

Pregnancy-related factors also play a critical role in the occurrence of PROM. For example, polyhydramnios, a condition characterized by an excess of amniotic fluid, increases the pressure on the fetal membranes [18]. This additional pressure stretches the membranes beyond their normal capacity, making them more vulnerable to rupture. The increased volume of fluid also heightens the risk of sudden pressure changes within the uterus, which can further stress the membranes and lead to premature rupture.

Multiple pregnancies, such as twins or triplets, also increase the likelihood of PROM. With more than one fetus, the physical strain on the uterus and membranes increases due to the added size and weight. The presence of additional amniotic fluid and fetal movement further stretches the membranes, increasing the chances of a rupture. These factors combine to put greater mechanical stress on the membranes, leading to a higher incidence of PROM in pregnancies involving multiple fetuses.

Certain medical procedures during pregnancy can inadvertently raise the risk of PROM. Procedures like amniocentesis, where amniotic fluid is sampled, carry the risk of puncturing or weakening the membranes [19]. Similarly, the placement of cerclage stitches to close the cervix can sometimes irritate the membranes, increasing the likelihood of rupture. Although these procedures are often necessary, they introduce additional risks that can lead to premature membrane rupture if complications arise.

Finally, trauma to the abdomen, such as from a fall or car accident, can dramatically increase the risk of PROM. Physical injuries can directly damage the amniotic sac or cause a sudden spike in intrauterine pressure, leading to rupture. Even relatively minor trauma can be dangerous, especially in the later stages of pregnancy when the membranes are already under significant strain from the growing fetus and the increasing volume of amniotic fluid. These traumatic events can cause unexpected membrane rupture, compounding the risks already present in the pregnancy [20].

Duration of PROM and Its Impact on Neonatal Outcomes

The time between membrane rupture and delivery is a crucial factor in determining neonatal outcomes, particularly in cases where the rupture occurs preterm. The longer the interval between PROM and delivery, the higher the risk of complications for both the mother and the baby. One of the primary concerns following PROM is the increased risk of infection. Once the amniotic sac ruptures, the sterile environment that previously protected the fetus is compromised, allowing bacteria from the vaginal canal to enter the uterus. This can result in serious maternal infections such as chorioamnionitis, which is an infection of the fetal membranes, and endometritis, an infection of the uterine lining after delivery [21]. For the neonate, the risk of infections such as neonatal sepsis becomes significant. This condition occurs when bacteria invade the infant's bloodstream, leading to a systemic infection. Preterm babies are especially vulnerable because their immune systems are underdeveloped, making them more susceptible to bacteria like group B Streptococcus (GBS) and Escherichia coli (E. coli).

Another infection-related risk for the neonate is pneumonia, which can develop from the aspiration of infected amniotic fluid or from bacteria transmitted during birth. In severe cases, meningitis, which is the infection of the membranes surrounding the brain and spinal cord, may occur, leading to potentially serious neurodevelopmental issues. The risk of infection rises significantly after 18 to 24 hours following membrane rupture. For this reason, when PROM is identified, close monitoring for signs of infection, such as fever, elevated white blood cell count, and fetal tachycardia, is essential [22].

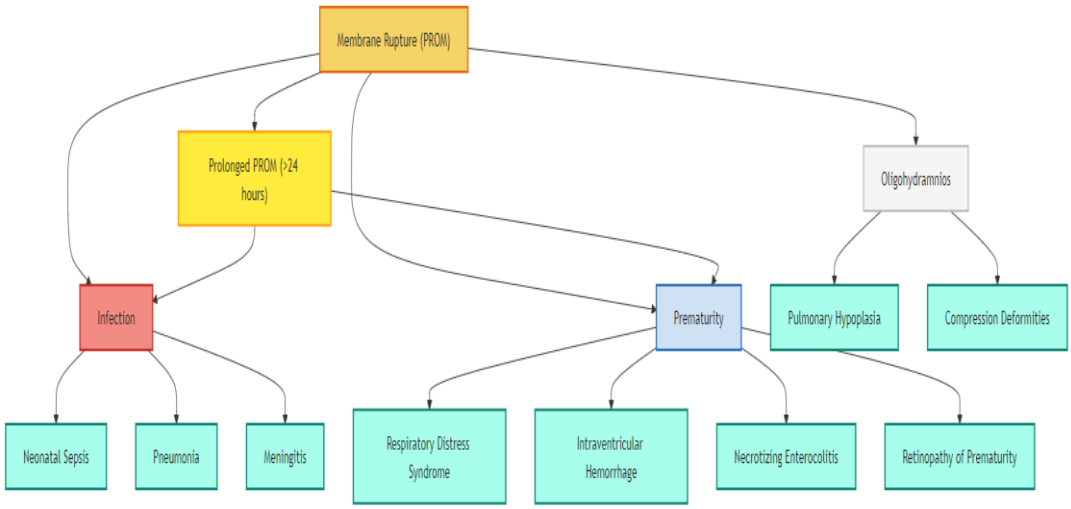

Figure 2: Complications of Prolonged Rupture of Membranes (PROM)

When PROM occurs early in gestation, it often results in complications related to prematurity. Preterm premature rupture of membranes (PPROM) is one of the leading causes of preterm birth, and babies born prematurely face a range of health challenges. Respiratory distress syndrome (RDS) is a common complication for preterm infants, caused by immature lungs that lack sufficient surfactant, a substance necessary for keeping the alveoli open. Without enough surfactant, breathing becomes difficult, and RDS can be life-threatening. Additionally, preterm infants are at increased risk for intraventricular hemorrhage (IVH), which is bleeding in the brain. This can lead to long-term neurodevelopmental issues, including motor and cognitive impairments [23].Another complication, necrotizing enterocolitis (NEC), affects the gastrointestinal system of preterm infants, causing inflammation and tissue death in the intestines. Premature infants are also at risk of developing retinopathy of prematurity (ROP), a condition involving abnormal development of blood vessels in the retina, which can result in blindness in severe cases.

Prolonged rupture of membranes can also lead to oligohydramnios, a condition characterized by a reduced volume of amniotic fluid. Amniotic fluid plays a vital role in the fetus's lung development and acts as a cushion, protecting the fetus from pressure. When oligohydramnios occurs, it can lead to pulmonary hypoplasia, where the lungs do not develop properly due to the lack of fluid, causing severe breathing

problems after birth. Additionally, low levels of amniotic fluid can result in physical deformities, such as clubfoot or cranial molding, caused by the compression of the fetus in the womb.

When PROM extends beyond 24 hours, the risks associated with the condition increase significantly. Infection becomes a major concern during this period, as prolonged exposure to the external environment raises the likelihood of both maternal and neonatal infections [24]. Neonatal early-onset sepsis, a potentially life-threatening infection, is a serious complication of prolonged PROM. While delivering the baby may sometimes be necessary to prevent infections and other complications, preterm delivery introduces additional risks related to the baby's immaturity, particularly if delivery occurs before the lungs have fully developed.

Pathophysiology of PROM

Premature Rupture of Membranes (PROM) involves complex interactions of several biological and mechanical processes, ultimately resulting in the rupture of fetal membranes before labor begins. When PROM occurs before 37 weeks of gestation, it is referred to as Preterm PROM (PPROM). The pathophysiology of PROM is multifaceted, involving structural changes in the membranes, inflammatory processes, enzymatic activity, and several other risk factors that contribute to premature membrane rupture [25].

The fetal membranes, composed mainly of collagen, elastin, and other extracellular matrix proteins, provide strength and elasticity to support the growing fetus. In PROM, increased activity of matrix metalloproteinases (MMPs) results in the degradation of collagen, particularly types I and III, which are essential for the tensile strength of the membrane. This leads to weakening of the membranes and an increased risk of rupture. Matrix metalloproteinases (MMPs) are normally balanced by tissue inhibitors of metalloproteinases (TIMPs), but in PROM, this balance is disrupted, leading to excessive collagen breakdown [26]. Additionally, oxidative stress also plays a role in the weakening of membranes by generating reactive oxygen species (ROS) that damage cellular components, including proteins, lipids, and nucleic acids. The increased oxidative stress further activates MMPs and contributes to tissue damage. Furthermore, increased apoptosis, or programmed cell death, within

the amniotic membrane reduces cell density and compromises its integrity. Cellular senescence, a state characterized by permanent growth arrest and the secretion of inflammatory mediators, is also observed in PROM and contributes to an increased risk of rupture by further promoting MMP production.

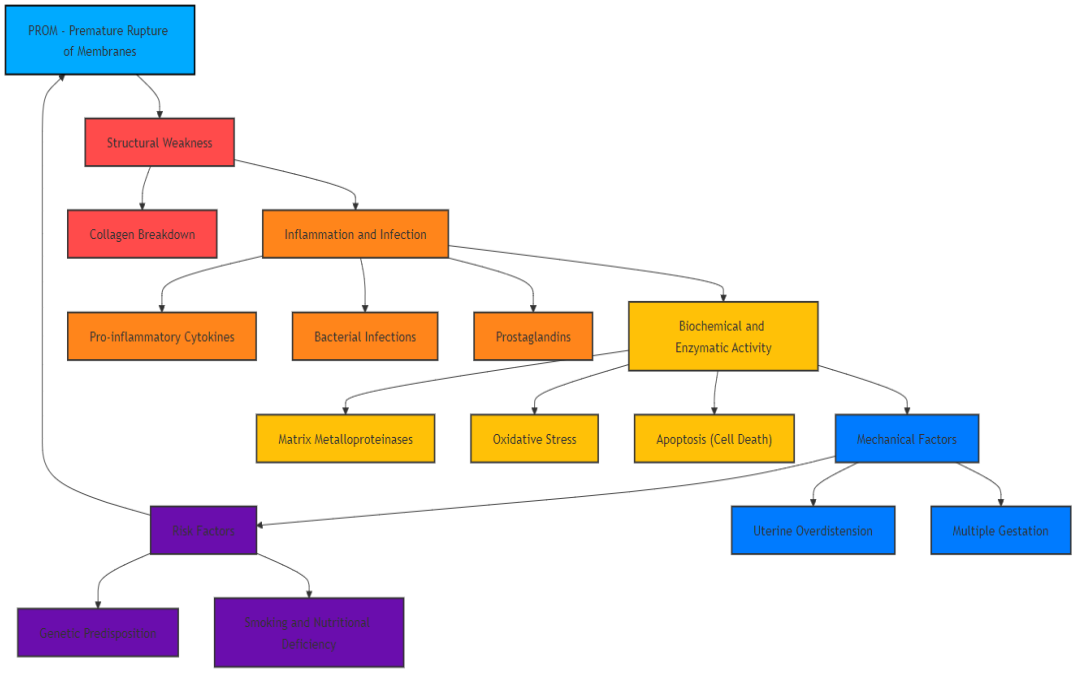

Figure 3: Pathophysiology of Premature Rupture of Membranes (PROM)

Another significant factor in the pathophysiology of PROM is infection and inflammation. Chorioamnionitis, or inflammation of the fetal membranes caused by microbial infection, is a common contributor to PROM. Bacterial pathogens such as *E. coli*, *Ureaplasma urealyticum*, *Mycoplasma hominis*, and Group B Streptococcus can ascend from the vagina into the uterus, leading to an inflammatory response. The release of pro-inflammatory cytokines such as IL-1, IL-6, and TNF-alpha activates enzymes that degrade the extracellular matrix, compromising membrane strength. The activation of Toll-like receptors (TLRs) in response to microbial invasion results in further cytokine production, amplifying the inflammatory response. The inflammatory cascade also increases prostaglandin production, which contributes to uterine contractions and further weakens the fetal membranes [27].

Mechanical factors also play a crucial role in PROM. Uterine overdistension due to conditions such as polyhydramnios or multiple gestations can lead to excessive physical stress on the fetal membranes, predisposing them to rupture. Overdistension causes focal areas of membrane weakening, leading to increased susceptibility to rupture under pressure. These focal weak spots are typically characterized by reduced collagen content and increased enzyme activity, making them vulnerable to mechanical stress. Genetic and environmental factors also contribute to the risk of PROM. Some individuals may have a genetic predisposition to PROM due to variations in genes involved in collagen synthesis, extracellular matrix regulation, or inflammatory pathways [28]. Polymorphisms in genes that encode MMPs or inflammatory cytokines, such as IL-6, can increase the likelihood of membrane rupture. Nutritional deficiencies, particularly in vitamin C, which is essential for collagen synthesis, can also play a role in weakening the membranes. Vitamin C is a cofactor for prolyl hydroxylase, an enzyme critical for collagen production, and deficiency impairs collagen synthesis. Trace element deficiencies, such as copper and zinc, may also negatively affect collagen cross-linking, further increasing the risk of PROM. Cigarette smoking is another contributing factor; toxic compounds in cigarette smoke, such as nicotine, impair collagen synthesis and promote oxidative stress, thereby weakening the fetal membranes.

Previous surgical procedures or trauma to the cervix can increase the risk of PROM. Procedures such as cervical cerclage or dilation and curettage can physically disrupt the fetal membranes, leading to a higher likelihood of premature rupture. Mechanical trauma to the membranes during these procedures can also contribute to membrane fragility. Additionally, cervical insufficiency—a condition in which the cervix fails to provide adequate support for the growing fetus—can lead to increased pressure on the membranes and subsequent rupture. An incompetent cervix may dilate prematurely, allowing ascending infection and increased membrane exposure to inflammatory processes, which further compromises membrane strength.

The pathophysiology of PROM is driven by multiple interconnected mechanisms, including structural degradation, infection, inflammation, mechanical forces, genetic predisposition, nutritional deficiencies, and external factors such as smoking [29]. Collagen degradation, oxidative stress, apoptosis, and inflammation weaken the

integrity of the fetal membranes, increasing the likelihood of rupture before labor begins. Understanding these mechanisms helps in the development of strategies for the prevention and clinical management of PROM, reducing the risks associated with preterm delivery and associated complications.

Amniotic fluid loss and its implications for fetal growth and lung development

Amniotic fluid plays a crucial role in the development and protection of the fetus throughout pregnancy. It creates a cushioning environment, enabling fetal movement for musculoskeletal development, and facilitates the exchange of nutrients and waste between the fetus and the mother. One of its most critical functions, however, is its role in fetal lung development. When there is a loss of amniotic fluid, known as oligohydramnios, the fetus faces significant risks, including growth restriction and compromised lung development [30]. This loss can prevent the fetus from having the necessary space and fluid to move freely, which is essential for musculoskeletal development. Additionally, the diminished volume of fluid can lead to intrauterine growth restriction (IUGR), where the fetus fails to grow at the expected rate.

Lung development is particularly dependent on amniotic fluid. In normal conditions, the fetus breathes in amniotic fluid, which aids in expanding the lung tissues, allowing for proper alveolar development and preparing the lungs for respiration after birth. When amniotic fluid is lost, this process is disrupted, often leading to pulmonary hypoplasia, a condition where the lungs remain underdeveloped. This condition is life-threatening, as it severely compromises the newborn's ability to breathe independently once born. Studies on animals have shown that a reduction in amniotic fluid at critical developmental stages can significantly impair lung formation and function. For example, research on rat fetuses demonstrated that the absence of sufficient amniotic fluid caused both pulmonary hypoplasia and growth retardation, highlighting the fluid's vital role in fetal lung maturation [31].

Oligohydramnios can occur due to several factors, including premature rupture of membranes or placental insufficiency. When this condition develops, especially during the early stages of pregnancy, it restricts lung development by reducing the amount of fluid the fetus inhales. The lack of fluid impedes the full expansion of the alveoli, the tiny air sacs in the lungs responsible for oxygen exchange, thus impairing

respiratory function post-birth. Additionally, prolonged periods of low amniotic fluid volume can result in severe growth retardation, and the fetus may suffer from significant respiratory complications after birth.

Amniotic fluid is also integral to the secretion of lung liquid, a specialized fluid produced by the fetal lungs. This liquid maintains the internal lung pressure, promoting the development of the lung tissues [32]. When the volume of amniotic fluid decreases, the pressure required for lung expansion diminishes, which in turn inhibits lung growth. This dynamic is critical, particularly during the second trimester, when lung formation is at a crucial stage. Without adequate amniotic fluid, the lungs may fail to mature properly, leading to long-term respiratory issues after birth.

In pregnancies complicated by chronic oligohydramnios, long-term health consequences for the newborn are more likely. Pulmonary hypoplasia is just one outcome; these infants are often born prematurely and face an increased risk of chronic lung diseases, as well as difficulties in breathing. Furthermore, oligohydramnios heightens the likelihood of preterm labor, which introduces additional challenges, such as underdeveloped organs and systems, including the lungs. Clinical cases have shown that chronic leakage of amniotic fluid significantly raises the risk of neonatal complications related to pulmonary hypoplasia, underscoring the importance of early intervention in pregnancies affected by this condition [33].

Monitoring the volume of amniotic fluid is therefore an essential aspect of prenatal care. By assessing the fluid levels, healthcare providers can identify early warning signs of fetal distress or underdevelopment. This information is particularly valuable in preventing respiratory complications post-birth. Adequate management and timely intervention in cases of oligohydramnios can help mitigate some of the more severe outcomes related to lung underdevelopment and other associated risks.

The importance of amniotic fluid for fetal growth and lung development cannot be overstated. Its loss presents significant risks to fetal well-being, particularly in the form of growth restriction and pulmonary hypoplasia [34]. The complex interplay between amniotic fluid levels and lung development highlights the need for

continuous monitoring and early detection of oligohydramnios, ensuring that interventions can be made to support better neonatal outcomes.

Inflammatory response in PROM and fetal exposure to pathogens

Prelabor rupture of membranes (PROM) occurs when the amniotic sac breaks before the onset of labor, leading to increased risk of fetal exposure to pathogens. This exposure can induce an inflammatory response in both the mother and fetus, which may have significant clinical consequences. When PROM takes place, the amniotic cavity becomes vulnerable to pathogens from the vaginal canal and external environment, increasing the likelihood of ascending bacterial infections.

Pathogens, such as bacteria, can cross into the amniotic fluid, activating an inflammatory response in the mother [35]. The maternal immune system recognizes the presence of these pathogens and responds by recruiting immune cells, including neutrophils, and releasing pro-inflammatory cytokines such as IL-1, IL-6, and TNF-α. This inflammatory response can result in chorioamnionitis, an infection of the amniotic membranes and placenta, which poses risks to both the mother and fetus.

The fetus can also mount its own immune response, referred to as Fetal Inflammatory Response Syndrome (FIRS). In FIRS, fetal immune cells like monocytes and neutrophils release cytokines into circulation, resulting in elevated levels of IL-6 in fetal blood or amniotic fluid. This fetal inflammatory response is associated with a range of adverse outcomes, including preterm birth, neonatal sepsis, cerebral palsy, and other neurodevelopmental complications.

Certain pathogens are commonly implicated in PROM-related infections, such as *Ureaplasma urealyticum*, *Mycoplasma hominis*, *Escherichia coli*, and group B streptococcus [36]. These pathogens can contribute to adverse fetal outcomes in several ways. Inflammatory processes can trigger the production of prostaglandins, leading to uterine contractions and preterm labor. Additionally, inflammation causes the release of matrix metalloproteinases (MMPs), which weaken the fetal membranes, increasing the risk of preterm birth.

The inflammatory environment in PROM can also impact fetal development and lead to complications such as respiratory distress, intraventricular hemorrhage, and increased susceptibility to sepsis. Pro-inflammatory cytokines can have direct neurotoxic effects on the developing fetal brain, increasing the risk of long-term neurodevelopmental disorders such as cerebral palsy.

To manage PROM and mitigate risks, several strategies are employed. Antenatal corticosteroids are often given to promote fetal lung maturity in cases of preterm PROM, reducing the risk of respiratory complications [37]. Antibiotic therapy is also used to reduce the risk of ascending infection and to prolong the latency period of pregnancy, allowing the fetus additional time to mature. Careful monitoring is essential to detect any signs of infection or fetal distress, and early delivery may be necessary if chorioamnionitis develops. Understanding the role of the inflammatory response in PROM is crucial to guiding effective interventions and minimizing risks to both mother and fetus.

Clinical Classification and Management of PROM

Prelabor Rupture of Membranes (PROM) occurs when the fetal membranes break before labor starts. It can be classified by how long it takes for labor to begin after the membranes rupture, as well as by the baby's gestational age. The duration of the rupture is often used to categorize PROM. When it lasts less than 24 hours, it is considered early PROM. If it persists for 24 to 48 hours, it is classified as prolonged PROM, while a rupture lasting more than 48 hours substantially raises the risk of complications, including infections in both the mother and baby [38].

The management of PROM is highly individualized, depending on factors such as the duration of rupture, gestational age, and any indications of complications like infection. The primary goal is to protect the health of both the mother and baby while reducing the risk of adverse outcomes. Generally, there are two management strategies: expectant management and early delivery. Expectant management is where labor is awaited naturally while closely monitoring the mother and baby. This approach is often selected if the gestational age is preterm and no signs of infection or fetal compromise are present. However, if there are indications of infection or fetal

distress, early delivery is pursued. When PROM occurs at term (37 weeks or more), labor is generally induced within 24 hours to lower the risk of infection.

The use of antibiotics is a crucial component of PROM management. They are given as a preventive measure when PROM occurs without labor, especially if the rupture lasts more than 18 hours [39]. This reduces the risk of maternal and neonatal infections, including Group B Streptococcus. In cases of preterm PROM (PPROM), antibiotics are particularly helpful as they can extend the latency period, which is the time between membrane rupture and delivery, ultimately lowering neonatal risks.

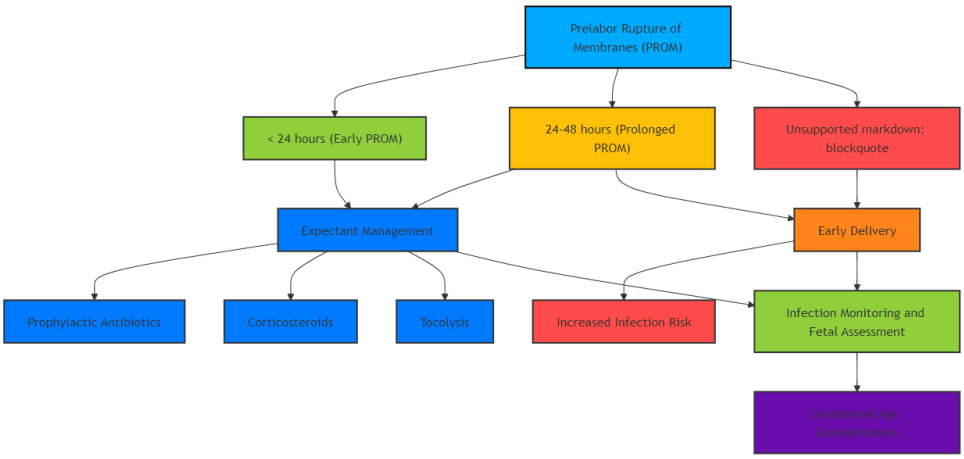

Figure 4: Clinical Classification and Management of PROM

Corticosteroids are also a key aspect of managing PROM, particularly for preterm PROM between 24 and 34 weeks of pregnancy. They are given to help speed up fetal lung development, which decreases the risk of complications like respiratory distress syndrome and intraventricular hemorrhage. Usually, a single course of corticosteroids is administered, with careful attention to the timing of delivery.

In some cases, medications called tocolytics may be used to delay labor for a short period to allow corticosteroids to take effect or to facilitate transfer to a facility that is better equipped to handle preterm births [40]. Tocolysis is typically considered for pregnancies between 24 and 34 weeks, provided that there are no signs of infection or other complications. Tocolytics are not used if there is any evidence of maternal infection or other issues that necessitate immediate delivery.

Monitoring is a critical part of managing PROM, especially to detect signs of infection as early as possible. Signs such as maternal fever, uterine tenderness, and fetal tachycardia are monitored, along with laboratory markers like increased white blood cell counts or elevated C-reactive protein. Fetal wellbeing is assessed with continuous or intermittent heart rate monitoring, and any signs of fetal distress may prompt immediate intervention.

The approach to managing PROM also depends on gestational age. When PROM happens at or after 37 weeks, delivery within 24 hours is often recommended to decrease the likelihood of infection. If PROM occurs between 34 and 36 weeks, early delivery is generally considered the best option, as it balances the risk of infection with that of prematurity [41]. For pregnancies between 24 and 33 weeks, the preferred course is often expectant management, aimed at safely extending the pregnancy as long as possible to improve neonatal outcomes. This typically involves using antibiotics, corticosteroids, and sometimes tocolytics, PROM management aims to balance the risks of infection and preterm birth while ensuring the health of both mother and baby. Close monitoring and timely intervention are essential to achieving the best outcomes for both.

Neonatal Outcomes Associated with PROM Duration

1. Respiratory Outcomes

Increased risk of respiratory distress syndrome (RDS) with prolonged PROM

Preterm rupture of membranes (PROM) is a serious complication in pregnancy, especially when it occurs early. One of the major concerns following PROM is the heightened risk of respiratory complications in newborns, particularly **respiratory distress syndrome (RDS)**. This condition is caused by the insufficient production of surfactant, a substance crucial for keeping the alveoli in the lungs open for effective gas exchange. In preterm infants, the lack of adequate surfactant leads to difficulty in breathing, making RDS one of the most critical challenges in neonatal care following PROM [42].

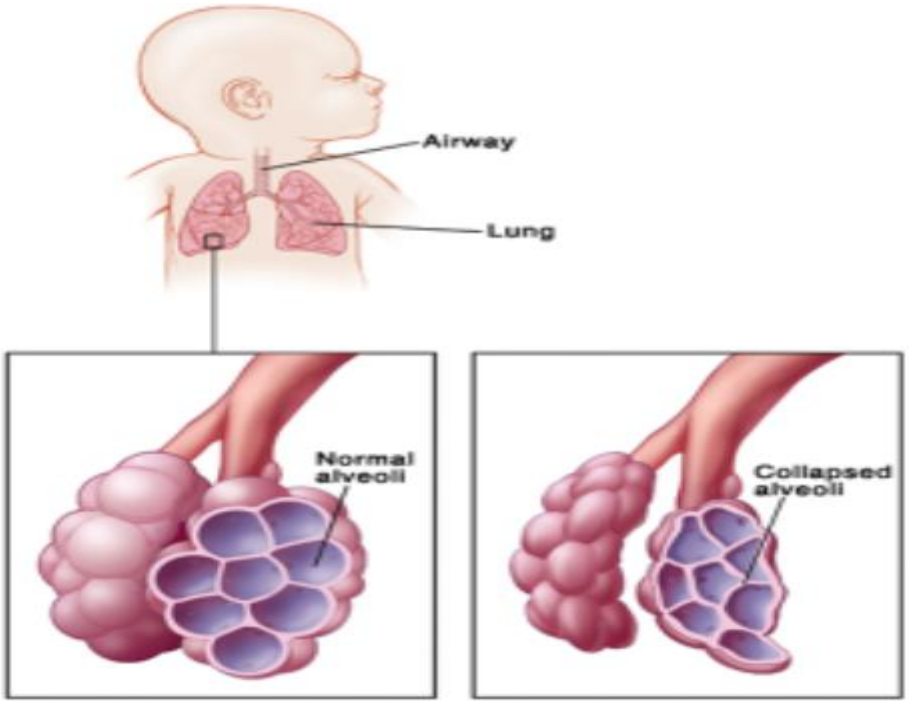

Figure 5: respiratory distress syndrome (RDS)

(Source: https://www.bravebeginnings.org/respiratory-distress-in-infants-and-adults/)

When PROM is prolonged, meaning the membranes have been ruptured for more than 18 to 24 hours before delivery, the risk of RDS increases significantly. Several factors contribute to this heightened risk. One such factor is the risk of infection. The longer the fetal membranes remain ruptured, the greater the chance of intrauterine infection, such as chorioamnionitis. Infections within the uterus trigger inflammatory responses that can hinder fetal lung development and decrease surfactant production, leading to a greater likelihood of RDS. The inflammatory damage to the fetal lung tissue can make it more difficult for the newborn to breathe effectively after birth [43].

Another factor increasing the risk of RDS in cases of prolonged PROM is the immaturity of the fetal lungs. PROM often leads to preterm birth, and infants born before 34 weeks of gestation are particularly vulnerable due to underdeveloped lungs. Preterm lungs do not produce enough surfactant, and without this essential substance, the infant's lungs are unable to expand properly, leading to respiratory distress. This

makes early delivery in cases of PROM a difficult decision, as the benefits of avoiding infection must be weighed against the risks of lung immaturity.

Oligohydramnios, or low levels of amniotic fluid, is another complication that can arise from prolonged PROM and further increases the risk of RDS [44]. Amniotic fluid plays a vital role in fetal lung development by allowing the lungs to expand within the uterus. Reduced amniotic fluid can lead to compression of the fetal thorax, impairing lung development and increasing the chance that the newborn will experience breathing difficulties after birth.

Managing prolonged PROM requires careful consideration of the timing of delivery to minimize both the risk of infection and the risk of RDS in the newborn. While early delivery may reduce the likelihood of infections, premature infants are at high risk of RDS due to their immature lungs. Interventions such as administering corticosteroids to accelerate lung development are often employed to improve outcomes for these infants. Respiratory Distress Syndrome remains a major concern in neonatal care following preterm birth due to prolonged PROM, and careful monitoring and timely interventions are essential to reducing its impact [45].

Role of Corticosteroids in Improving Neonatal Lung Function

Corticosteroids play a crucial role in enhancing neonatal lung function, particularly in the context of preterm births. Administering corticosteroids to pregnant women at risk of delivering prematurely has become a well-established intervention aimed at accelerating fetal lung maturity and reducing the risk of respiratory distress syndrome (RDS) in infants. This intervention is especially beneficial for infants born between 24 and 34 weeks of gestation, as their lungs are often underdeveloped and not capable of producing sufficient surfactant on their own.

The two corticosteroids most commonly used for this purpose are betamethasone and dexamethasone. These medications are typically given to pregnant women in cases of threatened preterm birth to promote lung development in the fetus [46]. One of the key benefits of corticosteroids is their ability to enhance surfactant production in the fetal lungs. Surfactant is essential for reducing surface tension within the alveoli, preventing their collapse, and enabling efficient gas exchange. By boosting surfactant

production, corticosteroids significantly improve the neonate's ability to breathe independently after birth.

Corticosteroids also contribute to the overall structural development of the lungs, improving lung compliance, which means the lungs can expand more easily. This increased compliance reduces the need for mechanical ventilation and oxygen therapy, which are often required for preterm infants with immature lungs. Studies have consistently shown that the use of corticosteroids in pregnancies at risk of preterm delivery greatly decreases the incidence and severity of RDS. Additionally, these medications lower the risk of other respiratory complications, such as bronchopulmonary dysplasia (BPD) and transient tachypnea of the newborn (TTN) [47].

Beyond the benefits to respiratory health, corticosteroids have been associated with reductions in overall neonatal mortality and the risk of other serious conditions like intraventricular haemorrhage and necrotizing enterocolitis. This broad range of positive outcomes makes corticosteroid administration a critical intervention in preterm births.

The standard regimen for corticosteroid administration involves giving two doses over a 24- to 48-hour period. In some cases, when preterm delivery occurs later than expected or if there is a prolonged delay, repeat or rescue courses of corticosteroids may be considered [48]. However, the benefits of multiple courses are still a subject of ongoing debate. Corticosteroids remain a cornerstone of care for pregnancies complicated by preterm labor or prolonged preterm rupture of membranes. They have been shown to significantly reduce the risk of RDS and improve neonatal respiratory outcomes, even when there is a risk of infection or inflammation in the uterus.

2. Neonatal Sepsis

Correlation between the duration of PROM and incidence of neonatal infections

The relationship between the duration of Prolonged Rupture of Membranes (PROM) and the risk of neonatal infections is a critical issue in both obstetrics and neonatology. As the time between membrane rupture and delivery increases, the risk

of neonatal infections becomes more significant. Once the rupture extends beyond 18 hours, the likelihood of infection, including conditions like early-onset sepsis, pneumonia, and meningitis, increases markedly. This prolonged exposure allows bacteria from the maternal genital tract to ascend into the amniotic fluid, exposing the fetus to potentially harmful pathogens.

The mechanism behind this correlation primarily involves bacterial invasion from the vagina into the amniotic cavity, a risk that increases as the time of exposure extends. Pathogens such as Group B Streptococcus (GBS) and Escherichia coli are among the most commonly implicated organisms [49]. The longer the rupture persists, the greater the chance that these or other bacteria will colonize the amniotic fluid, increasing the risk of neonatal sepsis. Research has shown that for every additional six hours that the membranes remain ruptured, the risk of neonatal sepsis increases significantly, reflecting the growing opportunity for bacterial infection.

Studies have provided statistical support for this correlation. Neonates born after membrane rupture lasting more than 18 hours have a much higher likelihood of developing infections compared to those whose exposure to PROM is shorter. In many cases, the risk of neonatal sepsis can be up to 1.5 to 2.5 times higher when PROM exceeds 18 hours, and further increases as the duration extends. The risk of infection is even more pronounced in cases of preterm PROM (PPROM), where the immaturity of the infant's immune system combined with prolonged exposure further elevates the risk of infection, there is a clear connection between the duration of PROM and the incidence of neonatal infections. The longer the interval between rupture and delivery, the greater the risk of bacterial colonization and infection, particularly sepsis. This highlights the importance of timely medical interventions to reduce the duration of PROM and mitigate infection risks, especially when the rupture lasts beyond 18 hours [50].

Early-onset neonatal sepsis and its relationship to maternal PROM

Early-Onset Neonatal Sepsis (EONS) is a serious infection occurring in newborns within the first 72 hours of life, and in some cases, within the first week. This condition can lead to severe complications such as respiratory failure, organ dysfunction, and even death if not treated promptly. One of the significant risk factors

for EONS is Premature Rupture of Membranes (PROM), which occurs when the amniotic sac breaks before the onset of labor. PROM increases the likelihood of infection by exposing the fetus to bacteria from the vaginal flora, which can ascend into the amniotic cavity and cause infection.

When PROM is prolonged—lasting more than 18 to 24 hours—the risk of neonatal sepsis rises considerably. This is because bacteria like Group B Streptococcus (GBS) and Escherichia coli (E. coli), which are common in the genital tract, have more time to move into the amniotic fluid, potentially leading to infections such as chorioamnionitis [51]. Chorioamnionitis is an inflammation of the membranes and amniotic fluid that can significantly increase the risk of sepsis in the newborn. If the mother develops this condition, the baby is at a much higher risk of infection due to exposure to these bacteria.

Newborns exposed to PROM, particularly if it is prolonged or complicated by infection, often show signs of illness shortly after birth. These may include difficulty breathing, lethargy, poor feeding, temperature instability, and abnormal blood test results, such as elevated or decreased white blood cell counts. In these cases, blood cultures and other laboratory tests are used to determine whether the newborn has sepsis, and treatment is started as soon as possible to prevent severe complications [52].

To reduce the risk of EONS in cases of PROM, several preventive measures are taken. In mothers who test positive for GBS or who experience prolonged PROM, intrapartum antibiotic prophylaxis is often administered to reduce the likelihood of bacterial transmission to the newborn. Additionally, delivering the baby promptly after PROM, particularly in cases where infection is suspected, can lower the risk of the newborn developing sepsis. If PROM occurs close to term, labor may be induced to minimize the duration of membrane rupture and prevent infection.

For neonates at risk due to PROM, particularly those born to mothers with prolonged membrane rupture or chorioamnionitis, close monitoring and, in some cases, empirical antibiotic treatment are standard practices [53]. Newborns are observed for any signs of infection, and if necessary, antibiotics are administered while awaiting the results of blood cultures. These preventive and treatment strategies help mitigate

the risks associated with PROM and early-onset neonatal sepsis, ensuring that any infection is identified and treated early to prevent complications.

3. Neonatal Mortality and Morbidity

The duration of time between the rupture of membranes and delivery plays a critical role in determining the risks associated with PROM. When PROM lasts more than 24 hours, the risk of neonatal infections such as chorioamnionitis and sepsis increases significantly, contributing to higher mortality rates. In preterm cases, prolonged latency can give the fetus more time to mature, but it also raises the risk of infection, which can be life-threatening. Mortality is particularly high when PROM occurs at extremely early gestational ages, such as before 24 weeks, due to the underdevelopment of the fetus's organs. As gestational age increases, particularly beyond 32 weeks, the risk of mortality decreases, especially with the use of antibiotics and corticosteroids to manage infections and promote lung development [54].

Long-term morbidities are a major concern for infants exposed to prolonged PROM, particularly in cases of preterm birth. Neurological complications are common, with cerebral palsy being one of the most severe outcomes. This is often the result of factors such as infection, hypoxic-ischemic injury, and intraventricular hemorrhage, which are more likely to occur after prolonged PROM. Infants are also at higher risk of neurodevelopmental delays and cognitive impairments, especially when PROM occurs at very early gestational ages. Additionally, periventricular leukomalacia, a form of brain injury affecting white matter, can lead to motor impairments [55].

Respiratory complications are also prevalent in infants born after prolonged PROM, particularly in those born before 32 weeks. Bronchopulmonary dysplasia, a chronic lung condition, is a frequent outcome due to prematurity, infection, and the need for prolonged mechanical ventilation. Infections like sepsis and meningitis can lead to long-term health problems, including cognitive and developmental challenges [56]. Other potential complications include retinopathy of prematurity, which can cause vision problems, and hearing impairments linked to infections or oxygen deprivation during the latency period. Despite advances in neonatal care, the duration of PROM continues to be a critical factor in determining both immediate survival and long-term health outcomes, especially for preterm infants.

4. NICU Admission and Length of Stay

Prolonged PROM is known to be a significant factor in predicting NICU admission and extended hospital stays for newborns. One of the key reasons is the increased risk of infection. Prolonged PROM raises the likelihood of maternal and neonatal infections such as chorioamnionitis, which can lead to complications for the newborn. These infants often require close monitoring, antibiotics, and additional support, resulting in their admission to the NICU. Another major issue tied to prolonged PROM is preterm birth, which is strongly associated with NICU admissions. Babies born prematurely are more prone to complications like respiratory distress, thermoregulatory challenges, and feeding difficulties, all of which require specialized neonatal care [57].

The length of time that membranes remain ruptured can also have an impact on the baby's lung development. If PROM occurs at an earlier gestational age, there may not be sufficient time for the fetal lungs to mature fully, which can lead to respiratory distress syndrome (RDS). This often necessitates the use of mechanical ventilation or other forms of respiratory support, contributing to the need for NICU care. Furthermore, prolonged PROM is linked to an increased risk of sepsis, which requires extended courses of antibiotics for the newborn, further prolonging the length of hospitalization.

Premature babies, particularly those affected by PROM, are also at risk for additional complications that can extend their hospital stay [58]. Conditions such as intraventricular hemorrhage (IVH), necrotizing enterocolitis (NEC), and retinopathy of prematurity (ROP) can develop, further complicating the newborn's recovery and necessitating longer periods of care. The effects of prolonged PROM on lung development can also lead to the development of oligohydramnios, which exacerbates respiratory issues and the need for prolonged respiratory support, increasing the overall length of stay in the NICU.

Another common challenge for infants born after prolonged PROM is feeding and growth delays. These infants may struggle with independent feeding and maintaining weight gain, both of which are critical milestones for NICU discharge. As a result, they often remain in the NICU until these issues are resolved, adding to the length of

their hospitalization. Ultimately, factors such as gestational age at birth, infection risk, respiratory complications, and neurodevelopmental concerns all play a role in determining the duration of a newborn's NICU stay [59]. The earlier a baby is born due to PROM and the more severe the associated complications, the longer their hospital stay is likely to be , prolonged PROM is a strong predictor of NICU admission and extended hospitalization due to the complex interplay of infection risk, respiratory challenges, feeding difficulties, and other complications related to prematurity. These factors collectively contribute to a longer recovery process for affected newborns.

5. Other Complications

Necrotizing enterocolitis (NEC), intraventricular hemorrhage (IVH), and retinopathy of prematurity (ROP) are serious conditions that predominantly affect premature infants, particularly those born before 32 weeks of gestation. These complications are a significant cause of morbidity and mortality in neonatal intensive care units (NICUs) and require specialized care to manage their complex nature.

NEC is a potentially fatal gastrointestinal disorder that leads to inflammation and, in severe cases, the destruction of the intestinal tissues in preterm infants. Although the exact cause of NEC is not fully understood, it is thought to stem from a combination of factors, including immature intestinal development, bacterial infections, and an abnormal immune response [60]. Babies affected by NEC may show symptoms such as abdominal swelling, feeding intolerance, lethargy, and, in some cases, blood in the stool. Treatment often involves stopping oral feeds, administering antibiotics, and providing supportive care. In severe instances, surgical intervention is required to remove the damaged sections of the intestine. NEC can lead to long-term complications, such as short bowel syndrome, particularly if large portions of the intestine are removed.

IVH, another common complication in premature infants, refers to bleeding into the brain's ventricular system. This condition typically affects very preterm infants, especially those born before 30 weeks gestation. The fragile blood vessels in the brain of premature infants are particularly susceptible to rupturing due to fluctuations in blood pressure or oxygen levels. IVH is graded on a scale from I to IV, with the

higher grades indicating more severe bleeding and a greater likelihood of long-term neurological damage. Mild cases of IVH may resolve without intervention, but more severe cases can result in conditions like hydrocephalus, where excess fluid accumulates in the brain, requiring surgical procedures like shunting. The long-term effects of IVH can include developmental delays, cerebral palsy, and other neurological impairments [61].

ROP is an eye disease unique to premature infants, characterized by the abnormal growth of retinal blood vessels. This abnormal vessel development can sometimes lead to retinal detachment and, if untreated, blindness. The risk of ROP is higher in infants born extremely prematurely or those requiring oxygen therapy. Regular screening of high-risk infants is essential to detect ROP early, as treatment options such as laser therapy or injections of anti-VEGF medications can prevent progression to severe stages. While mild cases of ROP often resolve on their own, severe forms can result in lifelong visual impairments or blindness if not managed appropriately.

These complications, all closely linked to the challenges of prematurity and low birth weight, highlight the critical need for early detection and prompt intervention in preterm infants [62]. A coordinated, multidisciplinary approach is essential for managing these conditions and improving outcomes for this vulnerable population.

Maternal and Obstetric Factors Influencing Neonatal Outcomes

Maternal infections during pregnancy, particularly conditions like chorioamnionitis and intra-amniotic infections, can significantly affect neonatal outcomes. Chorioamnionitis, an infection that causes inflammation of the fetal membranes, is often linked to preterm birth, which in turn increases the risk of complications such as neonatal sepsis, respiratory distress syndrome (RDS), and cerebral palsy. The inflammatory response triggered by these infections can interfere with fetal lung development, leading to a higher likelihood of respiratory issues like bronchopulmonary dysplasia (BPD) in preterm infants [63]. Additionally, severe intra-amniotic infections can provoke fetal inflammatory response syndrome (FIRS), a condition associated with long-term neurodevelopmental problems, including brain white matter injury and cognitive impairments.

Decisions regarding when to deliver, especially in complicated pregnancies, have a profound impact on neonatal health. In some cases, early delivery becomes necessary, such as in pregnancies complicated by preeclampsia or fetal growth restriction. However, preterm birth, particularly before 28 weeks, is associated with serious risks, including RDS, necrotizing enterocolitis (NEC), and intraventricular hemorrhage (IVH), and can lead to long-term neurodevelopmental issues or even death. On the other hand, expectant management, where the pregnancy is extended to allow further fetal development, can sometimes improve neonatal outcomes [64]. However, it must be carefully managed to avoid increasing the risk of maternal complications or fetal distress, which could result in stillbirth or neonatal asphyxia. In certain low-risk pregnancies, recent evidence suggests that elective delivery at 39 weeks can reduce the likelihood of neonatal complications without increasing the rate of cesarean sections.

The use of antibiotics and corticosteroids during pregnancy plays a crucial role in managing risks for neonates. Administering antibiotics to mothers with preterm premature rupture of membranes (PPROM) or chorioamnionitis can significantly reduce the chances of neonatal sepsis by preventing bacterial transmission from mother to child. However, prolonged use of antibiotics has been shown to potentially disrupt the neonatal gut microbiome, which could increase the risk of NEC. Corticosteroids, such as betamethasone and dexamethasone, are widely used to accelerate fetal lung maturation in anticipation of preterm birth, substantially decreasing the likelihood of RDS, IVH, and NEC. Clinical studies have also demonstrated that antenatal corticosteroids improve long-term neurodevelopmental outcomes, particularly in infants born before 32 weeks [65]. While a single course of corticosteroids is generally recommended, repeat doses are sometimes used with caution to avoid potential side effects, such as fetal growth restriction, managing maternal infections, determining the timing of delivery, and administering antibiotics and corticosteroids are all critical factors in optimizing neonatal outcomes. Careful balancing of these factors is essential in reducing risks associated with preterm birth and improving long-term health for newborns, particularly those in high-risk pregnancies.

There are several studies have been conducted from time to time which are presented below

Sirak B & Mesfin E; 2014 found that premature rupture of membranes (PROM) is associated with significant maternal and neonatal morbidity and mortality, particularly in Ethiopia, where maternal mortality remains high. A retrospective study at Mizan-Aman General Hospital aimed to examine maternal and fetal outcomes in cases of term PROM, analyzing data from 4,525 women who gave birth between January 2011 and December 2013, including 185 diagnosed with PROM. The study found that 22.2% of women and 33.5% of neonates experienced unfavorable outcomes, with puerperal sepsis as a leading cause of maternal morbidity. Factors such as PROM duration over 12 hours, latency over 24 hours, rural residency, and birth weight under 2,500 g were linked to adverse outcomes. The findings underscore the need for improved obstetric and medical care to prevent complications. [66].

Seliem W & Sultan AM;2018 aimed to determine the frequency of early-onset neonatal sepsis in newborns with varying durations of preterm premature rupture of membranes (PPROM) in the Neonatal Intensive Care Unit of Combined Military Hospital, Lahore, from November 2009 to November 2010. The study included neonates from singleton pregnancies complicated by PPROM, with delivery between 30 and 36 weeks of gestation. Of the 164 newborns, sepsis was clinically suspected in 25%, with 22% showing elevated C-reactive protein levels. The frequency of neonatal sepsis was significantly higher in cases with longer durations of membrane rupture ($p < 0.001$). The study concluded that PPROM is a key risk factor for early-onset neonatal sepsis, with a sepsis frequency of 22% [67].

Lorthe E et.al;2017 aimed to assess the impact of latency duration on survival, survival without severe morbidity, and early-onset sepsis in infants born after preterm premature rupture of membranes (PPROM) at 24-32 weeks' gestation. Based on data from the Etude Épidémiologique sur les Petits Âges Gestationnels 2 cohort in France, 702 singletons delivered after PPROM were analyzed. Latency duration was categorized into four periods: 12 hours to 2 days, 3-7 days, 8-14 days, and >14 days. Survival rates were 93.5%, survival without severe morbidity was 85.4%, and early-onset sepsis occurred in 3.4% of infants. While a crude association suggested that

prolonged latency improved survival, this effect disappeared when adjusting for gestational age at birth. Prolonged latency duration did not significantly impact survival without severe morbidity or the risk of early-onset sepsis. The study concluded that, at a given gestational age, prolonged latency after PPROM does not worsen neonatal outcomes [68].

Assefa NE et.al;2018 found that the incidence of premature rupture of membranes (PROM) ranges from 5% to 10% of all deliveries and poses significant risks to both mother and neonate, including infections, respiratory distress, and death. In Ethiopia, risk factors for PROM are not well studied, prompting this research to identify such factors among pregnant women in public hospitals in Mekelle city, Tigray. This unmatched case-control study, conducted from February to April 2016, involved 240 participants (160 controls and 80 cases). Data were collected through structured interviews and checklists. Multivariable logistic regression revealed that a history of abortion, PROM, cesarean section, and abnormal vaginal discharge were significant predictors of PROM. The study underscores the importance of early detection and treatment of genitourinary infections to mitigate the risks of PROM. [69].

Nayot D et.al;2012 aimed to identify factors associated with latency duration to birth after preterm premature rupture of membranes (PPROM) and its impact on neonatal outcomes. Data were collected on 1,535 singleton pregnancies with PPROM from a perinatal database at a tertiary care center. Latency was categorized as ⩽72 hours versus >72 hours. The percentage of women with latency >72 hours decreased from 67% in very preterm (25-28 weeks gestation) to 10% in late preterm (33-36 weeks gestation). Shorter latency (⩽72 hours) was linked to pregnancy-induced hypertension and lower birth weight, while longer latency (>72 hours) was associated with steroid administration and clinical chorioamnionitis. PPROM before 32 weeks with latency ⩽72 hours was linked to a two-fold higher risk of severe neonatal morbidity, while PPROM between 29-34 weeks with latency ⩽72 hours was associated with moderate neonatal morbidity. Longer latency periods (>72 hours) were linked to better neonatal outcomes, particularly up to 32 weeks for severe morbidity and up to 34 weeks for moderate morbidity [70].

Günes A et.al; 2022 aimed to identify significant predictors of neonatal infection in infants born to women with term premature rupture of membranes (PROM). Using multivariate analysis of data from the Term PROM Study, which compared labor induction with expectant management, several independent predictors were identified. The most significant predictors were clinical chorioamnionitis (odds ratio 5.89) and positive maternal group B streptococcal status (odds ratio 3.08). Other factors included a high number of vaginal digital examinations (odds ratio 2.37), longer durations from membrane rupture to active labor (24 to <48 hours, odds ratio 1.97; ≥48 hours, odds ratio 2.25), and maternal antibiotic use before delivery (odds ratio 1.63). The study concludes that chorioamnionitis and group B streptococcal colonization are the most important predictors of neonatal infection in infants born after term PROM [71].

Pugni L et.al;2016 examined the outcomes of neonates born to women with preterm premature rupture of membranes (PPROM) between 24 and 34 weeks of gestation, comparing those with and without a diagnosis of clinical chorioamnionitis. Among 1,153 patients diagnosed with PPROM, 29% were diagnosed with chorioamnionitis. Neonates born to these mothers had significantly higher incidences (34.8%) of adverse outcomes, including low 5-minute Apgar scores, respiratory distress syndrome (RDS), necrotizing enterocolitis (NEC), intracranial hemorrhage (ICH), and pneumonia, compared to 22.9% in neonates born to mothers without chorioamnionitis ($p < 0.001$). The study concludes that chorioamnionitis in the context of PPROM is associated with a higher risk of adverse neonatal outcomes [72].

Sorano S et.al;2020 examined the natural history and prognosis of pregnancies complicated by preterm premature rupture of membranes (pPROM) near the limit of viability, specifically between 20 and 23 weeks of gestation. The study included 66 cases treated at a single hospital between April 2007 and December 2017. Of these, 81.1% (54 newborns) survived to discharge, though 77.8% of the survivors experienced severe morbidity at the time of discharge. Multivariate analysis revealed that later gestational age at pPROM and longer latency periods were significantly associated with survival without severe morbidity. Of 23 cases followed at 36 months, 34.8% exhibited developmental delays. The survival rate was higher than in previous

studies, but many survivors faced severe short-term morbidities, although more than half showed normal development by 36 months. [73].

Kibel M et.al; 2016 assessed the natural history and outcomes of pregnancies complicated by previable preterm premature rupture of membranes (PROM) between 20 and 23 weeks of gestation. Conducted at a tertiary referral center from 2004 to 2014, the study included 104 neonates after excluding those with terminated pregnancies or major fetal anomalies. The results showed that 49% of neonates survived to discharge, with 47.1% of survivors experiencing severe neonatal morbidity (e.g., bronchopulmonary dysplasia, severe neurological injury, or retinopathy). Long-term morbidity was found in 23.3% of surviving neonates, particularly among those who had previously experienced severe neonatal morbidity. Key factors associated with survival and survival without severe morbidity included a gestational age of 22 weeks or greater at the time of PROM and a latency period of more than 7 days. However, expectant management posed significant maternal risks, including placental abruption (17.3%) and sepsis (4.8%). The study concludes that while neonatal survival after previable PROM is nearly 50%, survivors face high risks of severe morbidity, and maternal complications are considerable. [74].

Tchirikov M et.aj;2018 discussed that premature rupture of membranes (PROM), defined as the rupture of membranes before labor, occurs in about 10% of pregnancies, with the majority happening at or beyond 37 weeks of gestation. Only 10% of cases occur before 37 weeks, which increases risks for both the mother and fetus. Preterm PROM is associated with complications such as premature delivery, maternal and fetal infections, oligohydramnios affecting fetal development, placental abruption, cord prolapse, and fetal malposition. Expectant management, the standard treatment since the 1970s, often leads to preterm birth, raising concerns about the appropriate gestational age to shift from expectant management to active intervention. This article reviews the natural history of PROM, outcomes of expectant management, and associated maternal and fetal risks, with a focus on preterm fetuses. [75].

Deutsch A et.al; 2010 evaluated maternal and perinatal outcomes in 118 patients with premature rupture of membranes (PROM) between 16 and 26 weeks of gestation. The mean gestational age at diagnosis was 23.1 weeks, with a latency period that varied widely and showed no correlation to gestational age at rupture. Nearly half (48%) delivered within 3 days, and 83% delivered within 2 weeks. Significant complications included one maternal death from sepsis, 39% of patients developing amnionitis, and 6.8% experiencing placental abruption. The mean gestational age at delivery was 24.7 weeks, with a perinatal mortality rate of 67.7%. The survival rate was significantly lower in pregnancies with PROM at or before 23 weeks (13.3%) compared to those at 24-26 weeks (50%). Among 34 of 40 surviving infants followed up to 36 months, 67% had intact survival, while 33% had developmental abnormalities. The study suggests that expectant management in cases of PROM before 23 weeks should be approached cautiously and is justified primarily in patients who are fully informed of the risks. [76].

Gomez-Lopez N et. al;2010 highlighted several key points in their review of patients with premature rupture of membranes (PROM). First, in most term pregnancies, labor begins within 24 hours of membrane rupture, but earlier gestations often experience longer delays. About 20% of infants born after PROM weigh less than 2,500 grams. Amnionitis poses a significant risk to both mother and infant, with an incidence ranging from 5% to 25%, correlating with the length of the latent period. Perinatal mortality is approximately 5%, and factors such as a prolonged latent period, amnionitis, low birth weight, and breech presentation increase this risk. Prophylactic antibiotics do not reduce the incidence of amnionitis or perinatal mortality but may decrease postpartum morbidity. Administering antibiotics to newborns from aseptic PROM cases has not been proven to reduce perinatal deaths. The review suggests a standard definition and diagnostic method for PROM, advises against routine vaginal examinations to reduce the risk of amnionitis, and proposes that optimal outcomes occur when the infant weighs over 2,000 grams and the latent period is under 24 hours without infection. A management plan is discussed based on these findings. [77].

Van Der Ham DP et.al;2012 aimed to determine the optimal gestational age for labor induction after premature rupture of membranes (PROM) between 32 and 36 weeks' gestation, based on neonatal outcomes. A retrospective review of 236 patients

managed expectantly showed that prolongation of pregnancy by a week or more was uncommon, especially after 34 weeks. Neonatal outcomes improved significantly at 34 weeks, with reduced neonatal length of stay and lower incidence of hyperbilirubinemia compared to earlier gestations. No perinatal deaths were reported. The study concludes that a notable reduction in neonatal morbidity occurs at 34 weeks, supporting labor induction at this gestational age, as expectant management offers limited benefits due to short latency periods [78].

Hackenhaar AA et.al;2014 tested the hypothesis that various risk factors, including infections during pregnancy, independently increase the risk of preterm premature rupture of membranes (PROM) and preterm delivery without PROM. Using a case-control design, women aged 15–45 who experienced preterm PROM, full-term PROM, or preterm delivery without PROM were compared to matched controls with full-term deliveries. The study found that intra-amniotic infection, urinary tract infections, and gonorrhea significantly increased the odds of preterm PROM by 6.0, 3.7, and 7.6 times, respectively, even after accounting for factors like cigarette smoke exposure, prior preterm or full-term PROM, and antepartum bleeding. Women with previous preterm PROM were 4.8 times more likely to have preterm births without PROM, with smoking and antepartum bleeding also increasing the risk. The study concludes that infections, even after adjusting for other risk factors, are strongly associated with an increased likelihood of preterm PROM [79].

Drassinower et. al;2015 in a study on Prolonged Latency of Preterm Premature Rupture of Membranes and Risk of Neonatal Sepsisconcluded that 15.5% of patients in the cohort had confirmed sepsis and Prolonged exposure to an intrauterine environment of PPROM does not increase the risk of neonatal sepsis; prolonged PPROM ≥4weeks was associated with decreased risk of neonatal sepsis. They also found that Neonatal sepsis was also significantly associated with earlier gestational age at rupture of membranes [80].

Vijay et. al;2016 in his study on study of neonatal morbidity in preterm premature rupture of membrane mother found that the mostcommon clinical manifestations were respiratory distress syndrome 18 cases (36%), septicemia 4 cases (8%), meningitis 1 case (2%) andpneumonia 1 case (2%) [81].

Alam et. al;2013 in a study on Neonatal sepsis following prolonged rupture of membranes found 4% cases with blood-culture proven bacterial sepsis within 72 hours of birth. Klebsiella pneumonia (29%) and Pseudomonas aeruginosa (24%) were the commonest isolates followed by group B Streptococcus (18%) and Escherichia coli (12%). [82]

Ilker Kahramanoglu et.al;2018 in a study on Preterm Premature Rupture of Membranes and Neonatal and Maternal Outcomesconcluded that the most common neonatal morbidities were respiratory distress syndrome (45.8%), hyperbilirubinemia (34.8%), sepsis (17.1%), and congenital pneumonia (9.3%). Neonatal mortality ratewas 6.7%. [83]

MATERIALS AND METHODS

MATERIALS AND METHODS

Study Design and Setting

The present study is a hospital-based cross-sectional study conducted at Santosh Hospital, Ghaziabad. The study aims to evaluate neonatal outcomes in relation to the duration of premature rupture of membranes (PROM) and to analyze associated maternal risk factors.

Study Population

The study includes all neonates born to mothers with PROM lasting between 18-24 hours, 24-72 hours, and more than 72 hours. The minimum required sample size is 369 women, divided equally into three categories:

- **Category A:** 18-24 hours (n=123)
- **Category B:** 24-72 hours (n=123)
- **Category C:** >72 hours (n=123)

Ethical Considerations

Ethical approval for the study was obtained from the Ethical Committee of Santosh Medical College and Hospital, Ghaziabad. Informed consent was secured from all participating mothers before data collection.

Inclusion Criteria

- Neonates born to mothers with PROM of 18-24 hours, 24-72 hours, and >72 hours duration.

Exclusion Criteria

- Outborn neonates.
- Mothers with antepartum hemorrhage, toxemia of pregnancy, or comorbidities other than infections.

- Neonates with major congenital anomalies.
- Mothers with PROM lasting <18 hours.
- Mothers who received antibiotics prior to delivery.

Data Collection

A structured clinical research form was used to record maternal and neonatal details, including:

- Maternal demographic characteristics.
- Medical and obstetric history.
- Duration of PROM.
- Mode of delivery.
- Neonatal birth weight, Apgar scores, and morbidity outcomes.

STATISTICAL ANALYSIS

Data were compiled in MS Excel and analyzed using SPSS version 28.0. Statistical tests used include:

- MANOVA (Multivariate Analysis of Variables): To assess the interaction between PROM duration and neonatal outcomes.
- Chi-square test: To determine categorical data significance.
- T-test: For continuous variable comparisons.
- P-value <0.05 was considered statistically significant.

Neonatal Outcome Measures

The primary neonatal outcome measures included:

- Neonatal sepsis
- Birth asphyxia
- Respiratory distress syndrome
- Intraventricular hemorrhage (IVH)
- Necrotizing enterocolitis (NEC)
- Mortality rates (Case Fatality Rate - CFR)

Results Overview

- Mean maternal age: 29.12 ± 5.83 years
- Mean neonatal birth weight: 3.25 ± 0.70 kg
- Case Fatality Rate:
    - 18-24 hrs PROM: 0.51%
    - 24-72 hrs PROM: 1.90%
    - >72 hrs PROM: 4.35%
- The Chi-square test indicated a significant association between prolonged PROM and neonatal complications (p<0.0001)

RESULTS

RESULT

TABLE 1 :- Distribution of maternal age among study participants

Age Group	Number	Percentage (%)
18-22yrs 11 months	119	32.2
23-27yrs 11 months	142	38.5
27-32yrs 11 months	72	19.5
>32yrs 11 months	36	9.8
Total	369	100
Mean ± SD	**29.12±5.83years**	

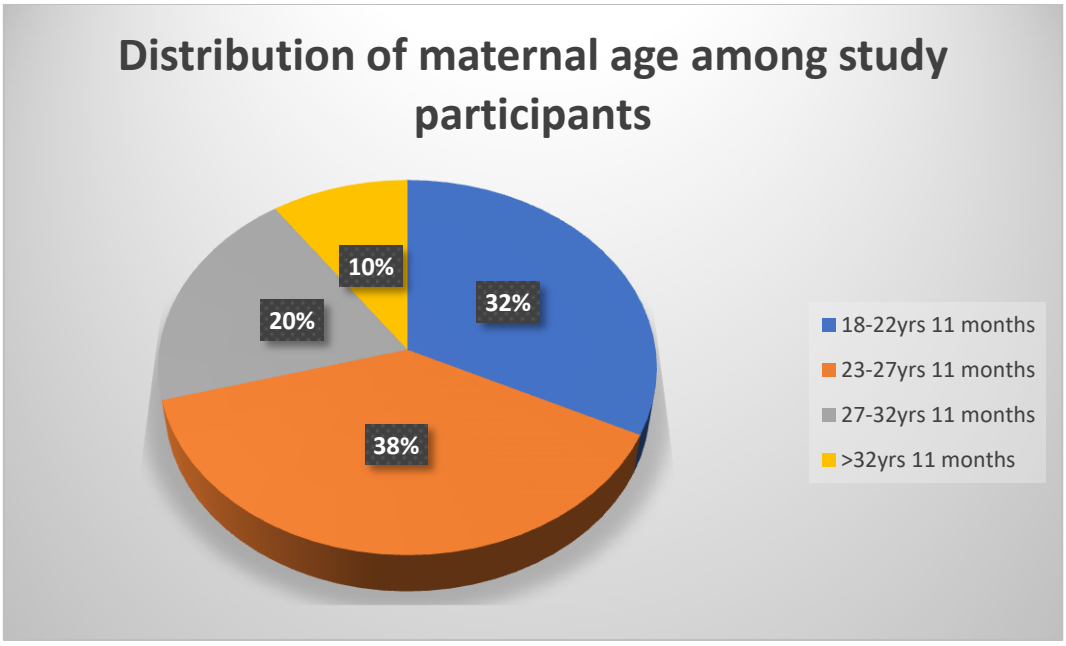

Figure 1: Distribution of maternal age among study participants

The age distribution of the study population shows that the majority of patients belong to the **23-27 years** age group (**38.5%**), followed by the **18-22 years** group (**32.2%**). The **27-32 years** group accounts for **19.5%**, while only **9.8%** of patients are older than **32 years**.

Table 2 : Line profile of mothers with rupture of membranes

Duration of ROM Group	Number	Percentage
18-24 hrs	195	52.85
24-72 hrs	105	28.46
>72 hrs	69	18.7
Total	369	100

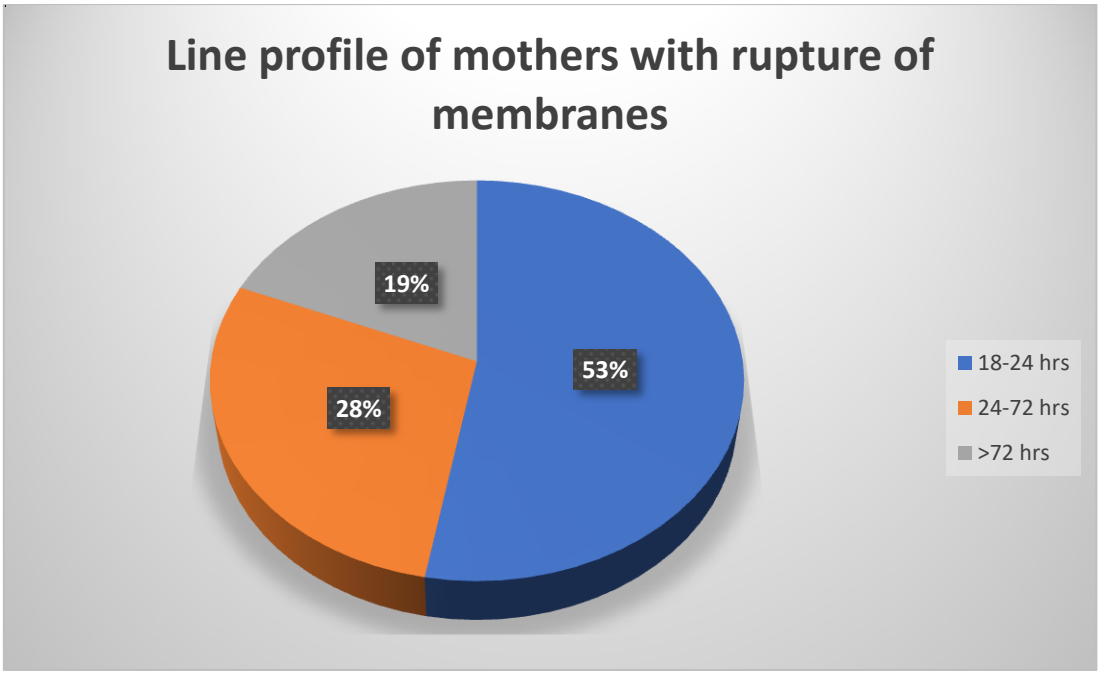

Figure 2: Line profile of mothers with rupture of membranes

The study included a total of 369 patients categorized based on the **duration of rupture of membranes (ROM)** into three groups: **18-24 hours, 24-72 hours, and more than 72 hours**. The majority of the patients were in the **18-24 hours group** (n=195, **52.8%**), followed by the **24-72 hours group** (n=105, **28.5%**) and the **>72 hours group** (n=69, **18.7%**).

Table 3: Maternal parity patterns among study participants

Parity	Number	Percentage (%)
Gravida 1	100	27.1
Gravida 2	81	22
Gravida 3	111	30.1
>Gravida 3	77	20.8

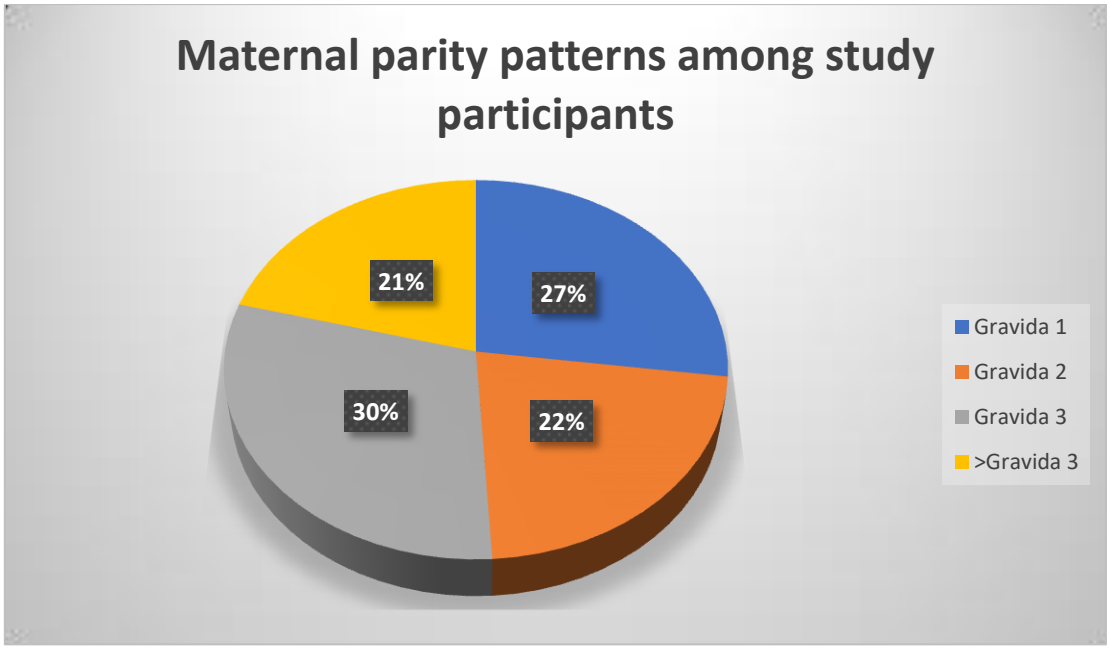

Figure 3: Maternal parity patterns among study participants

The **parity distribution** among the **369 patients** reveals that the most common group is **Gravida 3 (30.1%)**, followed by **Gravida 1 women (27.1%)**, while **Gravida 2 (22.0%)** and **more than Gravida 3 (20.9%)** constitute smaller proportions.

Table 4: Types of childbirth methods reported by participants

Delivery Type	Number	Percentage
Induced with Oxytocin	132	35.7
Spontaneous	123	33.3
Cesarean Section	114	30.9
Total	369	100

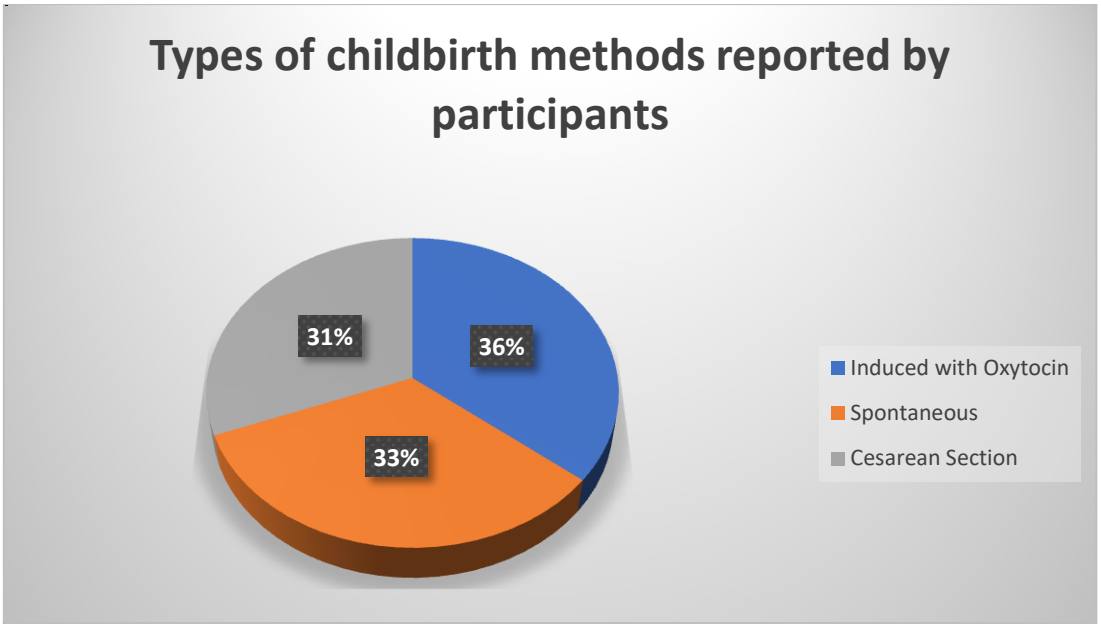

Figure 4: Types of childbirth methods reported by participants

The study included a total of **369 patients**, categorized based on their **mode of delivery**. 132 women (35.8%) delievered through induction with oxytocin, followed by 123 women (33.3%) delivered spontaneously and 114 women (30.9%) delievered by lower segment ceaserean section.

Table 5: Range of gestational periods at birth among participants

Gestational Age	Number	Percentage
<30weeks + 6 days	92	24.9
31-35weeks + 6 days	133	36
36-40weeks + 6 days	111	30.1
>41weeks + 6 days	33	8.9
Total	369	100

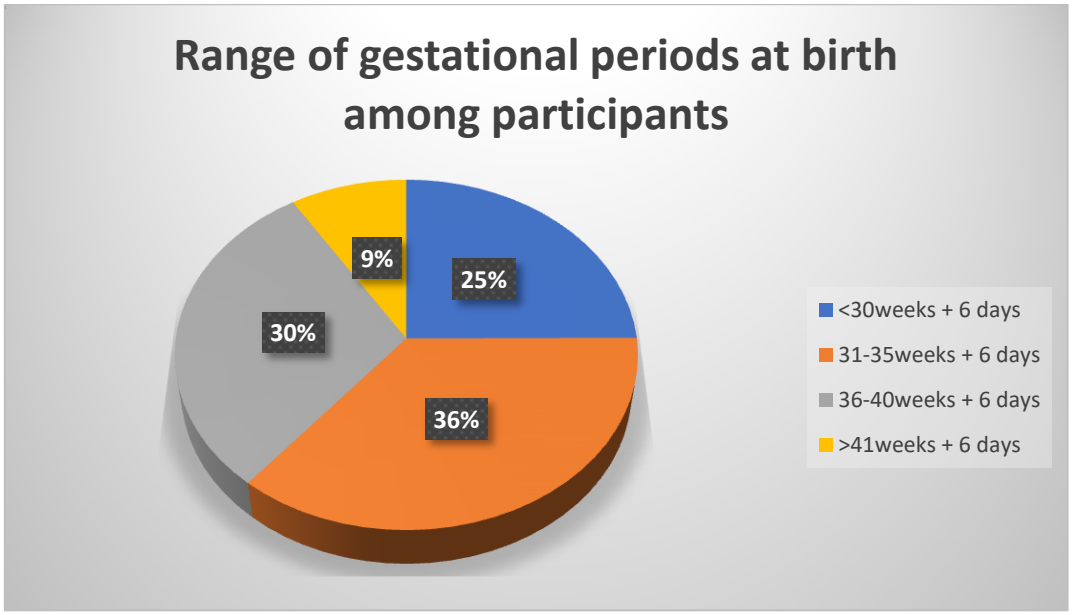

Figure 5: Range of gestational periods at birth among participants

The **gestational age distribution** among **369 patients** indicates that the **30-35 weeks** group is the most prevalent (**36.0%**), followed by the **35-40 weeks** group (**30.1%**). The **<30 weeks** category accounts for **24.9%**, while only **8.9%** of patients had a gestational age of **>40 weeks**.

Table 6: Specific causes for caesarean section among participants

Indication for Cesarean	Count	Percentage
Breech	41	35.9
Fetal Distress	39	34.2
Placenta Previa	34	29.8
Total	114	100

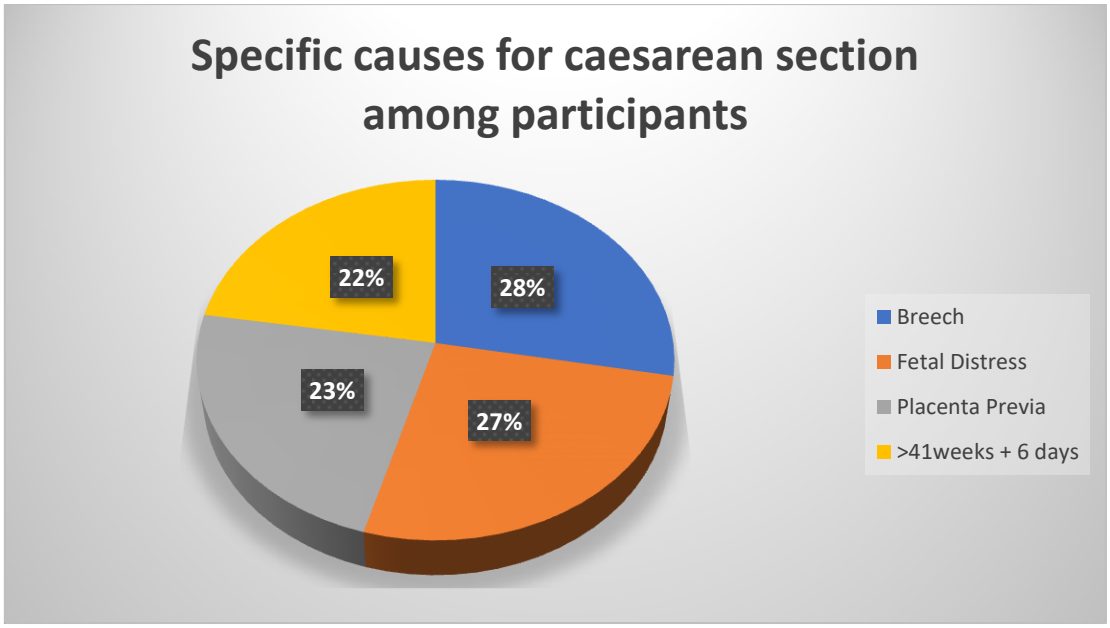

Figure 6: Specific causes for caesarean section among participants

The study included a total of **114 patients** who underwent **cesarean section** due to various indications. The most common indication was **Breech presentation** (**n=41, 36.0%**), followed by **Fetal Distress** (**n=39, 34.2%**) and **Placenta Previa** (**n=34, 29.8%**).

Table 7 : Outlining the frequency and types of health complications observed in newborns

Condition / gender	Male (n=168)	Female(n=201)	Number (n=369)	%
Congenital Abnormalities	1	2	3	0.8
Respiratory Distress	42	44	86	46.3
Neonatal Sepsis	70	76	146	79.4
Birth Asphyxia	40	70	110	48.5
IVH	15	8	23	6.2
NEC	0	1	1	0.3

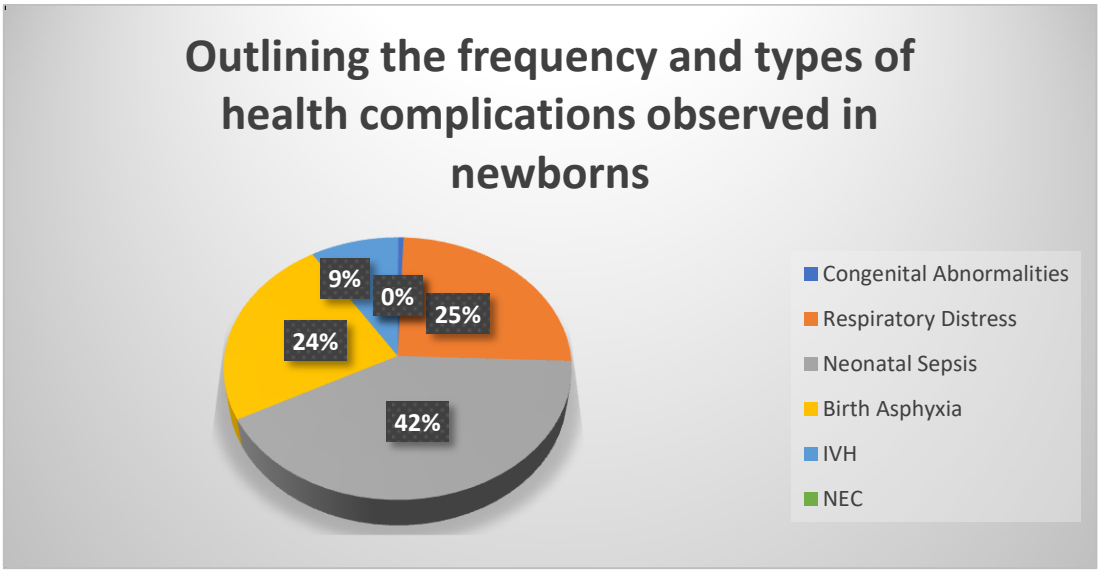

Figure 7: Outlining the frequency and types of health complications observed in newborns

The study analyzed the distribution of cases according to neonatal morbidity among **369 patients**. The most common neonatal condition was **Neonatal Sepsis**, observed in **293 cases (79.4%)**. **Birth Asphyxia** was present in **179 cases (48.5%)**, followed by **Respiratory Distress** in **171 cases (46.3%)**. Less frequently observed conditions included **IVH (Intraventricular Hemorrhage)** (**23 cases, 6.2%**), **Congenital Abnormalities** (**3 cases, 0.8%**), and **NEC (Necrotizing Enterocolitis)**, which was the least common condition, found in only **1 case (0.3%)**.

Table 8: Detailing the range and frequency of different weight categories at the time of delivery

Weight Category	number	Percentage
<1kg	92	24.9
1-2kg	125	33.9
2-3kg	130	35.2
3-4kg	18	4.9
>4kg	4	1.1
Total	369	100

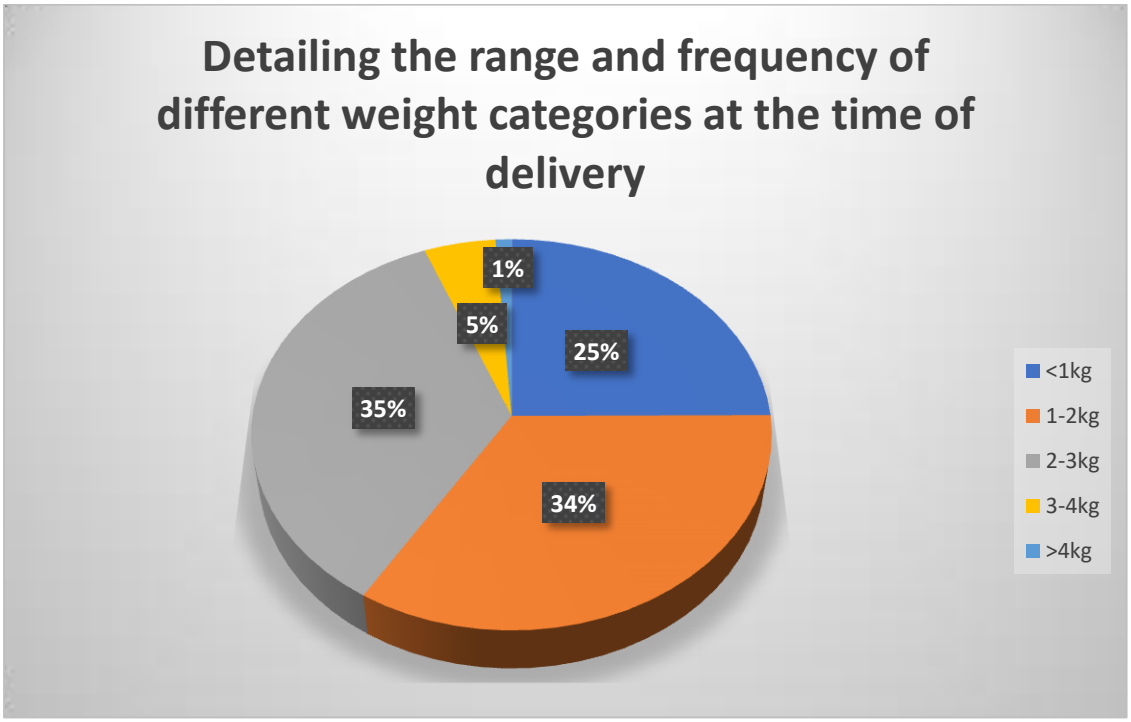

Figure 8: Detailing the range and frequency of different weight categories at the time of delivery

The birth weight distribution of 369 neonates indicates that the 2-3 kg group is the most prevalent (35.2%), followed closely by the 1-2 kg category (33.9%), while 24.9% of neonates weigh less than 1 kg. In contrast, only 4.9% of neonates fall in the 3-4 kg range, and just 1.1% weigh more than 4 kg.

Table 9: Ultrasound based observation and diagnosis during the study

Fetal USG	number	Percentage
Hyaline membrane disease	36	9.8
Meconium aspiration syndrome	23	6.2
Pneumonia	61	16.5
Transient tachypnea of newborn	42	11.4
NORMAL	180	48.8
OTHERS	27	7.3
Total	369	100.0

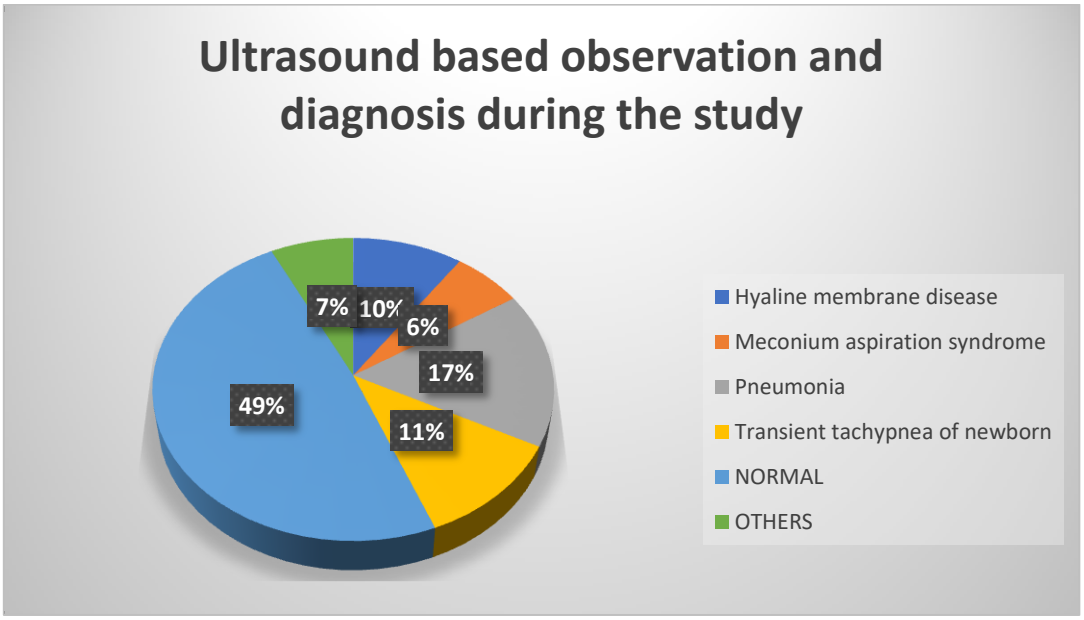

Figure 9: Ultrasound based observation and diagnosis during the study

The study analyzed **Fetal Cranial USG** findings in **369 newborns**, revealing a significant variation in distribution. The majority had **normal USG findings** (**n=180, 48.8%**). Among abnormal findings, **Respiratory Distress Syndrome (RDS)** was the most common (**n=61, 16.5%**), followed by **Transient Tachypnea of the Newborn (TTN)** (**n=42, 11.4%**), **Hyaline Membrane Disease (HMD)** (**n=36, 9.8%**), and **Meconium Aspiration Syndrome (MAS)** (**n=23, 6.2%**). Additionally, less frequent findings categorized as **'Others'** (**n=27, 7.3%**) included **IVH, Cystic Changes, Congenital Anomalies, and PVL**.

Table 10: Breakdown of neonatal morbidity: types and frequencies

Clinical manifestation	18-24hrs (n=195)		24-72hrs (n=105)		>72hrs (n=69)	
Congenital Abnormalities	1	0.005%	1	0.009%	1	0.014%
Respiratory Distress	87	0.446%	56	0.533%	28	0.405%
Neonatal Sepsis	160	0.820%	77	0.733%	56	0.811%
Birth Asphyxia	93	0.476%	52	0.495%	34	0.492%
IVH	14	0.071%	5	0.047%	4	0.057%
NEC	1	0.005%	0	0%	0	0%

The analysis of neonatal morbidity in relation to the duration of rupture of membranes (ROM) shows that **neonatal sepsis with p value of 0.186 (not significant)** was the most common complication, with **160 cases in the 18-24 hours group, 77 cases in the 24-72 hours group, and 56 cases in the >72 hours group**. **Respiratory distress with p value of 0.044 (significant association)** was also frequently observed, affecting **87 neonates in the 18-24 hours group, 56 in the 24-72 hours group, and 28 in the >72 hours group**. **Birth asphyxia with p value of 0.882 (not significant)** was another major morbidity, with **93 cases in the 18-24 hours category, 52 cases in the 24-72 hours category, and 34 cases in the >72 hours category**. Other complications, including **IVH and NEC**, were relatively less frequent across all ROM groups. These findings highlight the burden of neonatal complications associated with ROM, emphasizing the importance of timely monitoring and neonatal care to mitigate adverse outcomes.

Table 11: Overview of blood and clinical health indicators among participants

Parametrs	Mean ± standard deviation
Hb (gm/dL)	18.8 ±3.2
TLC (cells/mm3 x10^3)	15.2 ±4.5
Neutrophils (%)	59.8±6.2
Lymphocytes (%)	30.0±6.1
Monocytes (%)	4.9±2.1
Eosinophils (%)	1.5±0.5
Basophils (%)	0.5±0.5
CRP (mg/L)	9.8±5.5
Apgar 1min	4.5±3.0
Apgar 5min	6.9±1.4

The analysis of key hematological and clinical parameters of mothers reveals that the **mean hemoglobin (Hb) level** is approximately **18.85 mg/dL** with a standard deviation of **3.21 mg/dL**, indicating some variation among patients. The **total leukocyte count (TLC)** has a mean of **15,204 cells/mm³** with a standard deviation of **4,500 cells/mm³**, suggesting a broad range of values. Among the differential counts, **neutrophils** constitute an average of **59.78% (SD: 6.25%)**, while **lymphocytes** average **30.01% (SD: 6.06%)**, reflecting typical immune response patterns. **Monocytes** account for an average of **4.93% (SD: 2.08%)**, and further distribution of **eosinophils, basophils, and CRP levels** follows expected trends.

DISCUSSION

DISCUSSION

The study, titled "Outcome in Neonates Born to Mothers with Premature Rupture of Membrane of Varying Duration," examines the effects of PROM on neonatal health outcomes. The study was conducted at Santosh Hospital, Ghaziabad, using a cross-sectional study design. It included 369 mothers with PROM categorized into three groups based on the duration of membrane rupture: 18-24 hours, 24-72 hours, and more than 72 hours.

In present study, the age distribution of the study population shows that the majority of patients fall within the 23-27 years age group (38.5%), followed by the 18-22 years group (32.2%). The 27-32 years group comprises 19.5% of patients, while only 9.8% are older than 32 years. The mean age of the participants was 29.12±5.83 years. This distribution suggests that women in their early to late 20s represent the largest portion of the study population, which is consistent with general trends observed in obstetric studies. When comparing our study to previous research on the outcomes of neonates born to mothers with premature rupture of membranes (PROM) of varying duration, a study by **Endale T et.al; 2016** showed that younger maternal age (less than 25 years) was associated with higher rates of preterm birth and neonatal morbidity, particularly in cases of prolonged PPROM. In contrast, older maternal age (over 35 years) showed a higher incidence of complications like infection and uterine rupture, which also influenced neonatal outcomes[10] .

In present study, the distribution of the duration of rupture of membranes (ROM) among the 369 patients revealed that the majority (52.8%) had a rupture duration of 18-24 hours, followed by 24-72 hours (28.5%) and more than 72 hours (18.7%). This distribution provides insight into the frequency of prolonged membrane rupture within the cohort, with most patients experiencing a relatively shorter duration before labor. A comparison with findings from other studies, such as those by **Boskabadi H et.al; 2019** , indicates that a similar trend is observed, with the majority of women experiencing ROM for less than 24 hours. They found that neonates born after a rupture duration of 18-24 hours had lower rates of neonatal sepsis and respiratory distress compared to those whose membranes were ruptured for more than 72 hours, which correlates with our study's findings. Also highlighted that prolonged rupture

(>72 hours) significantly increased the incidence of neonatal infections and adverse respiratory outcomes [11].

In the conducted study, the distribution of parity among the 369 mothers revealed that G3 (30.1%) was the most common group, followed by nulliparous women (G1) at 27.1%. G2 mothers constituted 22.0%, while women with parity >G3 accounted for 20.8%. This suggests that women with a history of one or two prior pregnancies were more likely to experience premature rupture of membranes (PROM), with a notable proportion of nulliparous women also represented. Comparing our results with studies by **Lorthe E et.al; 2017** and Williams et al. (2018), a similar trend is observed, where multiparous women (G2 and G3) are often more prone to PPROM, although nulliparous women also experience it at significant rates. They reported that nulliparity is associated with an increased risk of PPROM, possibly due to factors such as cervical insufficiency. On the other hand, **Joyce SJ. in 2008** highlighted that multiparous women, especially those with more than two pregnancies, may face greater obstetric risks, including a higher likelihood of prolonged rupture and subsequent neonatal complications [12][13].

In current study, the distribution of delivery types among the 369 patients revealed that 35.7% of women were induced with oxytocin, followed by 33.3% who delivered spontaneously, and 30.9% who had a lower segment cesarean section (LSCS). This distribution suggests that induction of labor is a common approach in managing PROM, with spontaneous labor occurring in a significant proportion of cases. Cesarean delivery was the least common mode, likely due to obstetric factors such as fetal distress or other complications that required surgical intervention A comparison with studies by **Boskabadi H et.al; 2019** shows some similarities and differences in delivery mode following PROM. Boskabadi H et.al; found that induction with oxytocin was the most common method of delivery, particularly in women with prolonged rupture, similar to our findings. However, observed a higher cesarean section rate (40%) in cases of PPROM, suggesting a more frequent need for surgical intervention when labor was not progressing or when fetal compromise was suspected [11].

In present study, the distribution of gestational age among the 369 patients showed that the majority (36.0%) were in the 31-35 weeks group, followed by 30.1% in the 36-40 weeks group. The <30 weeks category accounted for 24.9%, while only 8.9% of patients delivered at >40 weeks gestation. This suggests that preterm deliveries were more common, with a significant proportion of women delivering between 31 and 35 weeks. When compared to studies by **Boskabadi H et.al; 2019** , our findings align with broader trends observed in cases of premature rupture of membranes (PROM). Boskabadi H et.al; reported that the majority of women with PROM had deliveries between 30 and 36 weeks, with a similar distribution observed in our study. **Blackmon LR, et.al; 1986** also found that gestational age at delivery in PROM cases tended to be skewed toward earlier weeks, with the highest rates of neonatal complications occurring in those born before 32 weeks. These studies support our observation that earlier gestational ages are more frequent in PROM cases, likely due to the increased risk of infection and premature labor [11][14].

In present study, the distribution of cesarean section indications among 114 patients revealed that the most common reason for surgical delivery was breech presentation (35.9%), followed by fetal distress (34.2%) and placenta previa (29.8%). These findings highlight the obstetric complications that often necessitate cesarean delivery in cases of premature rupture of membranes (PROM). When compared to studies by **Eslamian L et.al; 2002** , our findings are consistent with the broader trends observed in cesarean section indications in PROM cases. Eslamian L et.al; reported that breech presentation was the leading indication for cesarean delivery in PROM cases, similar to our study, due to the higher risk of complications associated with vaginal breech delivery. They also observed fetal distress as a common indication for cesarean section, which is consistent with our results, especially in cases of prolonged PROM, where fetal well-being is often compromised. Placenta previa, while less frequent, remains a significant cause of cesarean delivery in PROM, as noted in both our study and the study due to the increased risk of hemorrhage [15].

In present study, neonatal morbidity was most commonly associated with neonatal sepsis, observed in 79.4% of cases, followed by birth asphyxia (48.5%) and respiratory distress (46.3%). Intraventricular hemorrhage (IVH) was found in 6.2% of neonates, while congenital abnormalities and necrotizing enterocolitis (NEC) were

much less common, at 0.8% and 0.3%, respectively. The gender distribution revealed slightly higher incidences of neonatal sepsis and birth asphyxia in males, with females showing higher rates of respiratory distress. When comparing our findings with studies by **Pharande P et.al; 2017** , our results align with the common pattern of neonatal complications seen in preterm births due to PROM. Pharande P et.al; also found neonatal sepsis to be the most frequent morbidity, affecting 70% of their cohort, similar to our results. They also observed high rates of respiratory distress and birth asphyxia, reflecting the immaturity of the neonates' organs, especially in cases of PROM. Also reported IVH and NEC as less frequent but still significant in extremely preterm neonates, supporting the rarity of these conditions in our study [16].

In present study, the birth weight distribution of neonates showed that the majority (35.2%) were in the 2-3 kg range, followed by 33.9% in the 1-2 kg category. Neonates weighing less than 1 kg comprised 24.9%, while only 4.9% of neonates had a birth weight between 3-4 kg, and 1.1% exceeded 4 kg. These findings suggest that preterm neonates are more likely to have low birth weights, with a significant proportion weighing under 2 kg. Comparing our results with studies by **Pendse A et.al; 2021** , similar patterns of low birth weight are observed in neonates born after PROM. They reported that the majority of neonates born with PROM had birth weights in the 1-2 kg or 2-3 kg range, mirroring our study's findings. Also found that preterm neonates, particularly those born before 32 weeks of gestation, were more likely to have a birth weight under 2 kg, correlating with the increased risk of complications in these neonates. Our study highlights that despite varying PROM durations, low birth weight remains a predominant risk factor for neonatal morbidity [17].

In the conducted study examining ultrasound findings in 369 newborns, the majority (48.8%) exhibited normal fetal cranial ultrasound results. Among those with abnormal findings, Respiratory Distress Syndrome (RDS) was the most prevalent (16.5%), followed by Transient Tachypnea of the Newborn (TTN) at 11.4%. Hyaline Membrane Disease (HMD) and Meconium Aspiration Syndrome (MAS) were less common, with respective occurrences of 9.8% and 6.2%. Other less frequent findings, categorized under 'Others' (7.3%), included intraventricular hemorrhage (IVH), cystic changes, congenital anomalies, and periventricular leukomalacia (PVL). **González-Mesa E et.al; 2021** highlight the variation in fetal cranial ultrasound outcomes, with

normal results being the most frequent, yet various respiratory and neurological disorders also represented a significant proportion. In comparison, a study investigating neonates born to mothers with premature rupture of membranes (PROM) of varying durations found that neonatal outcomes were significantly affected by the duration of PROM. Newborns exposed to prolonged PROM had higher incidences of infections, respiratory distress, and other complications compared to those with a shorter duration of PROM, indicating the importance of timely intervention and monitoring [[18].

In the current study on neonatal morbidity distribution, clinical manifestations were analyzed at different time intervals post-birth: 18-24 hours, 24-72 hours, and >72 hours. The most frequent condition observed was neonatal sepsis, with 160 cases (0.820%) in the first 18-24 hours, followed by a decrease in the subsequent intervals. Respiratory distress was also prominent, especially within the first 18-24 hours, with 87 cases (0.446%). Birth asphyxia was noted in 93 (0.476%) neonates within the first 18-24 hours, and its occurrence remained relatively consistent across the time intervals. Less frequent conditions such as IVH and NEC were observed in much smaller numbers. Congenital abnormalities showed an extremely low incidence throughout all time periods, with just one case in each interval. In comparison, **Kurkinen-Räty M et.al; 1998** study on neonates born to mothers with premature rupture of membranes (PROM) revealed that prolonged PROM significantly increased the risk of neonatal morbidity. Neonates exposed to longer durations of PROM had higher incidences of respiratory distress, sepsis, and infections. These complications were more frequent in neonates born after prolonged PROM compared to those exposed to shorter PROM durations [19].

In conducted study on the distribution of hematological and clinical parameters in neonates, the mean hemoglobin (Hb) level was 18.8 ± 3.2 gm/dL, reflecting a relatively high level of hemoglobin. The total leukocyte count (TLC) averaged 15.2 ± 4.5 × 10^3 cells/mm^3, with neutrophils making up the majority (59.8%) of the white blood cells, followed by lymphocytes at 30.0%. The presence of other cell types, such as monocytes (4.9%), eosinophils (1.5%), and basophils (0.5%), was noted in lower percentages. C-reactive protein (CRP) levels had a mean of 9.8 ± 5.5 mg/L, indicating an inflammatory response. Apgar scores at 1 minute and 5 minutes were 4.5 ± 3.0 and

6.9 ± 1.4, respectively, reflecting a degree of initial distress, with improvement by the fifth minute. **Hincu MA et.al; 2024** studies on neonates born to mothers with premature rupture of membranes (PROM) found that prolonged PROM led to significantly altered hematological and clinical parameters. These neonates often showed elevated CRP levels, indicating increased infection risk, and had lower Apgar scores, which were associated with higher rates of neonatal sepsis, respiratory distress, and other complications [20].

SUMMARY

SUMMARY

SUMMARY

- The age distribution of the study population reveals that most patients are in the 23-27 years age group (38.5%), followed by those in the 18-22 years group (32.2%). The 27-32 years age group represents 19.5%, while only 9.8% of patients are older than 32 years.

- The study involved 369 patients, who were grouped based on the duration of rupture of membranes (ROM) into three categories: 18-24 hours, 24-72 hours, and more than 72 hours. The largest group was the 18-24 hours category (n=195, 52.8%), followed by the 24-72 hours group (n=105, 28.5%), and the more than 72 hours group (n=69, 18.7%).

- The study included 369 patients, classified according to their mode of delivery. A total of 132 women (35.8%) delivered through oxytocin induction, followed by 123 women (33.3%) who delivered spontaneously, and 114 women (30.9%) who underwent a lower segment cesarean section.

- The distribution of gestational age among the 369 patients reveals that the 30-35 weeks group is the largest (36.0%), followed by the 35-40 weeks group (30.1%). The <30 weeks group makes up 24.9%, while only 8.9% of patients were in the >40 weeks category.

- The study included 114 patients who underwent cesarean section for various indications. The most common reason was breech presentation (n=41, 36.0%), followed by fetal distress (n=39, 34.2%) and placenta previa (n=34, 29.8%).

- The study examined the distribution of neonatal morbidity among 369 patients. Neonatal sepsis was the most prevalent condition, affecting 293 cases (79.4%). Birth asphyxia was present in 179 cases (48.5%), followed by respiratory distress in 171 cases (46.3%). Less common conditions included intraventricular hemorrhage (IVH) in 23 cases (6.2%), congenital

abnormalities in 3 cases (0.8%), and necrotizing enterocolitis (NEC), which was the least frequent, occurring in only 1 case (0.3%).

- The birth weight distribution of the 369 neonates shows that the 2-3 kg group is the most dominant, comprising 35.2% of the total, followed by the 1-2 kg group at 33.9%. A significant portion, 24.9%, of neonates weigh less than 1 kg. In contrast, only 4.9% fall within the 3-4 kg range, while just 1.1% have a birth weight exceeding 4 kg.

- The study examined fetal cranial ultrasound (USG) findings in 369 newborns, showing notable variation in the results. The majority had normal USG findings (n=180, 48.8%). Among the abnormal findings, Respiratory Distress Syndrome (RDS) was the most prevalent (n=61, 16.5%), followed by Transient Tachypnea of the Newborn (TTN) (n=42, 11.4%), Hyaline Membrane Disease (HMD) (n=36, 9.8%), and Meconium Aspiration Syndrome (MAS) (n=23, 6.2%). Additionally, less common findings categorized as 'Others' (n=27, 7.3%) included conditions such as IVH, cystic changes, congenital anomalies, and periventricular leukomalacia (PVL).

- The analysis of neonatal morbidity in relation to the duration of rupture of membranes (ROM) reveals that neonatal sepsis, with a p-value of 0.186 (not significant), was the most common complication. It affected 160 neonates in the 18-24 hours group, 77 in the 24-72 hours group, and 56 in the >72 hours group. Respiratory distress, with a p-value of 0.044 (significant association), was also frequently observed, affecting 87 neonates in the 18-24 hours group, 56 in the 24-72 hours group, and 28 in the >72 hours group. Birth asphyxia, with a p-value of 0.882 (not significant), was another major morbidity, with 93 cases in the 18-24 hours group, 52 cases in the 24-72 hours group, and 34 cases in the >72 hours group. Other complications, such as IVH and NEC, were less frequent across all ROM categories. These findings underscore the prevalence of neonatal complications related to ROM, highlighting the need for careful monitoring and timely neonatal care to reduce adverse outcomes.

- The analysis of key hematological and clinical parameters in mothers shows that the mean hemoglobin (Hb) level is approximately 18.85 mg/dL, with a standard deviation of 3.21 mg/dL, indicating some variation among patients. The mean total leukocyte count (TLC) is 15,204 cells/mm^3, with a standard deviation of 4,500 cells/mm^3, suggesting a wide range of values. Among the differential counts, neutrophils average 59.78% (SD: 6.25%), while lymphocytes make up an average of 30.01% (SD: 6.06%), reflecting typical immune response patterns. Monocytes account for an average of 4.93% (SD: 2.08%), with the distribution of eosinophils, basophils, and CRP levels following expected trends.

CONCLUSION

CONCLUSION

The study highlights the significant impact of PROM duration on neonatal health. The findings confirm that prolonged PROM (>72 hours) is associated with a higher incidence of neonatal sepsis, respiratory distress, and birth asphyxia. The increasing case fatality rate with prolonged PROM further underscores the need for timely intervention and enhanced monitoring of both mothers and neonates.

Early identification of risk factors, including maternal infections, low socioeconomic status, and previous history of PROM, can help in the timely administration of antibiotics and corticosteroids to improve neonatal outcomes. The study also emphasizes the importance of prenatal care in reducing the risk of complications associated with PROM.

Limitations

1. **Single-Center Study**: The study was conducted at a single hospital, which may limit the generalizability of findings to other populations.
2. **Short Follow-up Period**: Long-term neonatal outcomes beyond the immediate postnatal period were not assessed.
3. **Lack of Detailed Infection Analysis**: The study did not analyze specific pathogens responsible for neonatal sepsis, which could provide more targeted intervention strategies.
4. **Limited Sample for Severe PROM Cases**: Although the study included 69 cases in the >72-hour category, a larger sample could strengthen statistical power and further validate findings.

Recommendations

1. **Routine Screening for Maternal Infections**: Early detection and treatment of maternal infections can reduce the incidence of neonatal sepsis.

2. **Early Hospitalization and Induction Protocols**: Timely labor induction in cases of prolonged PROM can help prevent adverse neonatal outcomes.

3. **Comprehensive Neonatal Monitoring**: Enhanced neonatal care, including early sepsis screening and respiratory support, should be implemented for at-risk neonates.

4. **Longitudinal Studies**: Future research should focus on long-term developmental outcomes in neonates born after PROM to assess potential neurodevelopmental impacts.

5. **Multicenter Studies**: Conducting similar studies across multiple hospitals can provide broader insights into regional variations in neonatal outcomes and healthcare practices.

In conclusion, this conducted study provides critical evidence on the neonatal risks associated with PROM and emphasizes the need for early intervention strategies. By improving prenatal care, infection control measures, and timely delivery planning, adverse neonatal outcomes associated with PROM can be significantly reduced, thereby improving neonatal survival rates and long-term health.

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ANNEXURES

PATIENT INFORMATION SHEET

Title: "Outcome in neonates born to mothers with Premature Rupture Of Membrane of varying duration."

Introduction: This statement describes the purpose, procedures, benefits, risks and discomforts of the study and your right to withdraw from the study at any point of time.

Study Procedure: Relevant clinical history, detailed examination, clinical assessment, and investigations will be done.

Confidentiality: Record of the patient's study participation will be kept confidential. Any publication of data will not identify patient by any means like name, photographs etc. By signing the consent form you authorize the sharing of your study related medical records to the regulatory authorities and the Institutional Ethical Committee.

Information regarding withdrawal: You have the right to withdraw from the study at any time during the study without any prejudice to you or your family's right to undergo treatment at Santosh Medical College and Hospital.

Contact for additional information: Any time during or after the study, you can obtain further information about the study from:

Name of Investigator	Name of Guide
Dr. Shubhangi Bansal	Dr. Alka agarwal
Post Graduate Resident	Dean & Professor
Department of Pediatrics	Department of Pediatrics
Santosh medical college and Hospital,	Santosh medical college and Hospital
Ghaziabad, U.P	Ghaziabad, U.P

रोगी सूचना पत्र

शीर्षक: "विभिन्न अवधि के लिए प्रीमैच्योर रप्चर ऑफ मेम्ब्रेन (PROM) से उत्पन्न माताओं के नवजातों में परिणाम।"

परिचय: यह वक्तव्य अध्ययन के उद्देश्य, प्रक्रियाओं, लाभों, जोखिमों और असुविधाओं का विवरण करता है और यह आपकी अधिकारिता है कि आप अध्ययन से किसी भी समय वापस ले सकते हैं।

अध्यान प्रक्रियाएँ: संबंधित चिकित्सा इतिहास, विस्तृत परीक्षा, नैदानिक मूल्यांकन और जांचें की जाएंगी।

गोपनीयता: रोगी के अध्ययन में भागीदारी का रिकॉर्ड गोपनीय रखा जाएगा। डेटा के किसी भी प्रकाशन में रोगी की पहचान नाम, फ़ोटो आदि के द्वारा नहीं की जाएगी। सहमति पत्र पर हस्ताक्षर करके आप अध्ययन से संबंधित चिकित्सा रिकॉर्ड को नियामक अधिकारियों और संस्थागत नैतिक समिति के साथ साझा करने की अनुमति प्रदान करते हैं।

अध्यान से वापसी के बारे में जानकारी: आपको अध्ययन से किसी भी समय वापस लेने का अधिकार है, इस प्रक्रिया से आपको या आपके परिवार को संतोष मेडिकल कॉलेज और अस्पताल में उपचार प्राप्त करने के अधिकार में कोई विघ्न नहीं आएगा।

अधिक जानकारी के लिए संपर्क: अध्ययन के दौरान या इसके बाद, आप निम्नलिखित से अध्ययन के बारे में और जानकारी प्राप्त कर सकते हैं:

अनुसंधानकर्ता का नाम:
डॉ. शुभांगी बंसल
पोस्ट ग्रेजुएट रेजिडेंट
बाल रोग विभाग
संतोष मेडिकल कॉलेज और अस्पताल
गाज़ियाबाद, उत्तर प्रदेश

मार्गदर्शक का नाम:
डॉ. अलका अग्रवाल
डीन और प्रोफेसर
बाल रोग विभाग
संतोष मेडिकल कॉलेज और अस्पताल
गाज़ियाबाद, उत्तर प्रदेश

CASE PERFORMA

STUDY CASE No.

A) Maternal detail

Name: MRD NO.

Address :

Age:

Date of admission: Date of discharge:

Date of delivery: Duration of stay:

LMP: POG:

Obs History:

Time of rupture: Type of delivery with outcome:

B) Neonatal detail

Gender: Weight:

APGAR: Abnormalities:

Investigation of neonate:

Hb-

TLC- DLC-

CRP- Blood culture-

Findings of ultrasound:

IVH HMD

NEC TTN

Sepsis Pneumonia

C) Neonatal outcome:

INFORMED CONSENT

Name of the patient: Age:

Address: Phone Number:

S.NO: Date:

I have been explained the details of the study entitled "OUTCOME IN NEONATES BORN TO MOTHERS WITH RUPTURE OF MEMBRANES OF VARYING DURATION" and my questions regarding the study have been answered to my satisfaction in a language understood by me.

1. I understand that I have the right to withdraw from the study at anytime and to decline to answer any particular question.

2. I understand that my participation in this study is confidential and that no material that could identify me will be used in the analysis and in any reports based on this study.

I hereby provide my consent in the study entitled "OUTCOME IN NEONATES BORN TO MOTHERS WITH RUPTURE OF MEMBRANES OF VARYING DURATION".

Signature/ thumb impression of the attendant

Signature/ thumb impression of witness

सूचित सहमति

रोगी का नाम: उम्र:

पता: फोन नंबर:

क्रम संख्या: तारीख:

मुझे अध्ययन का विवरण समझाया गया है जिसका शीर्षक है "विभिन्न अवधि के लिए रप्चर ऑफ मेम्ब्रेन (PROM) से उत्पन्न माताओं के नवजातों में परिणाम" और मेरे द्वारा पूछे गए सवालों के उत्तर मुझे मेरी समझ में आने वाली भाषा में दिए गए हैं।

1. मुझे समझ में आता है कि मुझे कभी भी अध्ययन से बाहर निकलने का और किसी विशेष प्रश्न का उत्तर देने से मना करने का अधिकार है।

2. मुझे समझ में आता है कि इस अध्ययन में मेरी भागीदारी गोपनीय है और कोई भी सामग्री जो मुझे पहचान सके, इस अध्ययन के विश्लेषण और रिपोर्टों में उपयोग नहीं की जाएगी।

मैं यहां "विभिन्न अवधि के लिए रप्चर ऑफ मेम्ब्रेन (PROM) से उत्पन्न माताओं के नवजातों में परिणाम" अध्ययन में अपनी सहमति देता/देती हूं।

अटेंडेंट के हस्ताक्षर/ अंगूठे का निशान

गवाह के हस्ताक्षर/ अंगूठे का निशान

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F. No. SU/R/2023/2489[35] Dated: 26.09.2023

Subject: Grant of Ethical Clearance for **Thesis Protocol** Topic **"Outcome in neonates born to mothers with premature rupture of membrane of varying duration",** submitted by **Dr. SHUBHANGI BANSAL,** MD/MS student of Batch 2022-2023, Department of **PAEDIATRICS.**

*****

With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"Outcome in neonates born to mothers with premature rupture of membrane of varying duration", Dr. SHUBHANGI BANSAL** is informed that the **Thesis Protocol** submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 17.07.2023 to 28.07.2023. The recommendations of the **Screening Committee** were considered in detail by the **Institutional Ethics Committee** in its meeting held on **23.09.2023** and the same was **approved** by the **Ethics Committee.**

He /She is informed accordingly for further necessary action.

[Dr. NARESH SHARMA]
REGISTRAR

SANTOSH "DEEMED TO BE UNIVERSITY" REGISTRAR GHAZIABAD, NCR DELHI

Dr. SHUBHANGI BANSAL
MD/MS student of batch 2022-2023
Department of PAEDIATRICS
Email: cutesshubhi@gmail.com

Copy to:

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2. The Dean, SMC&H
3. The Dean – Academics
4. The Dean – Research
5. The Director - IQAC
6. Dr. Alka Agrawal (Guide)
7. HOD of PAEDIATRICS

Dr.Shubhangi Bansal

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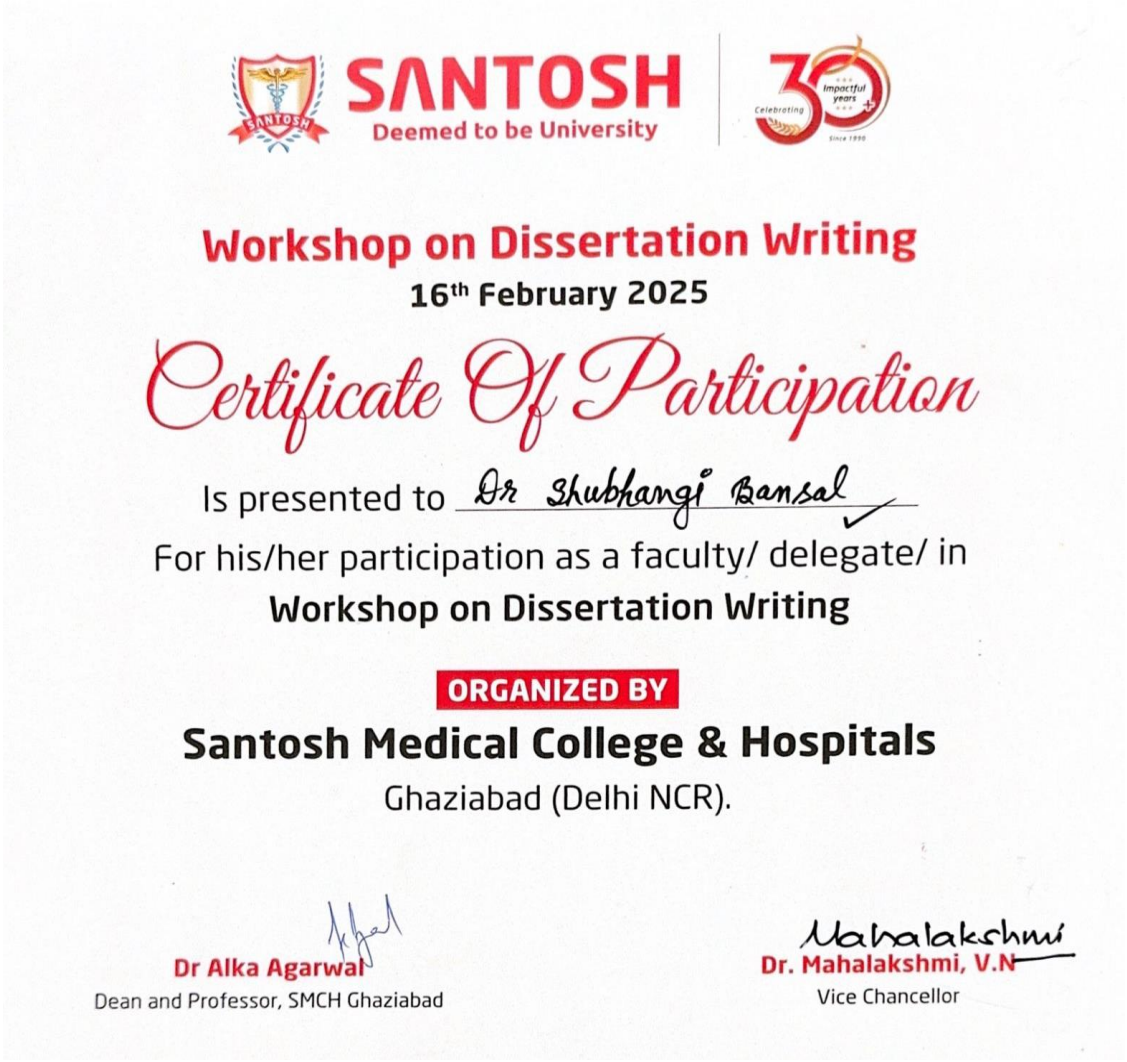

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Dr. Mahalakshmi, V.N
Vice Chancellor

MASTER CHART

MASTER CHART

S.No	Name	Age	Date of Admission	Date of Discharge	History	Duration of ROM	LMP	EDD	Obs History	Hb (mg/dL)	TLC (cells/mm3)	Neutrophils (%)	Lymphocytes (%)	Monocytes (%)	Eosinophils (%)	Basophils (%)	CRP (mg/L)	Apgar 1min	Apgar 5min	Congenital Abnormalities	Respiratory Distress	Neonatal Sepsis	Birth Asphyxia	IVH	NEC	Death	Cause of Death	Delivery Type	Indication for Cesarean	Blood Culture	Glucose	Fetal Cranial USG	Sex (M/F)	Weight (kg)	Gestational Age (weeks)
1	Patient_1	26	1/1/2024	1/13/2024	Yes	27	7/3/2023	4/8/2024	G3P2	17	12574	66	30	7	1	0	20	4	5	No	No	None	Yes	No	No	No		Spontaneous		Streptococcus	134	IVH 2	M	3.94	31
2	Patient_2	18	2/1/2024	2/14/2024	Yes	80	7/4/2023	4/9/2024	G4P3	24	11249	61	27	4	1	1	7.5	4	5	No	No	None	Yes	No	No	No		Induced with Oxytocin		Streptococcus	80	normal	M	2.51	33
3	Patient_3	24	3/1/2024	3/3/2024	Yes	43	8/23/2023	5/29/2024	G2P1	22	11673	70	31	8	1	0	15	8	8	No	Yes	Umbilical Sepsis	No	No	No	No		Cesarean Section	Placenta Previa	Negative	93	HMD	M	3.01	28
4	Patient_4	19	4/1/2024	4/4/2024	Yes	19	8/31/2023	6/6/2024	G2P1	14	11437	58	33	5	2	1	19	9	8	No	Yes	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	84	HMD with BPD	F	3.75	29
5	Patient_5	35	5/1/2024	8/1/2024	No	0	7/1/2023	4/6/2024	G2P1	19	28419	65	21	7	2	1	12.3	9	8	No	No	None	No	No	No	No		Induced with Oxytocin		Negative	79	Normal	M	4.18	40
6	Patient_6	32	6/1/2024	6/4/2024	Yes	28	7/15/2023	4/20/2024	G2P1	24	13165	68	28	6	2	0	12	1	5	No	Yes	Septicemia	Yes	No	No	No		Spontaneous		Negative	63	TTN	F	2.54	33
7	Patient_7	18	7/1/2024	7/3/2024	Yes	63	7/30/2023	5/5/2024	G4P3	17	11770	55	34	3	1	0	19	5	7	No	Yes	Pneumonia	Yes	No	No	No		Spontaneous		Negative	44	TTN	F	3.05	35
8	Patient_8	25	8/1/2024	11/1/2024	No	0	8/14/2023	5/20/2024	G2P1	18	11147	54	20	3	2	0	11.5	5	5	No	No	None	No	No	No	No		Spontaneous		Negative	40	NORMAL	M	2.51	32
9	Patient_9	29	9/1/2024	1/16/2024	No	0	8/29/2023	6/4/2024	G2P1	16	23105	70	32	6	1	0	17	4	5	No	No	None	No	No	No	No		Induced with Oxytocin		Negative	132	Normal	M	2.11	37
10	Patient_10	33	10/1/2024	10/13/2024	No	0	9/13/2023	6/19/2024	G1P0	14	14962	53	25	4	2	0	5	1	6	No	No	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Streptococcus	137	NORMAL	M	3.18	39
11	Patient_11	30	11/1/2024	11/3/2024	No	0	9/28/2023	7/4/2024	G1P0	23	15363	70	27	6	2	0	5	5	5	No	Yes	Conjunctivitis	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	81	IVH 1 WITH MAS	M	3.63	37
12	Patient_12	19	12/1/2024	12/4/2024	No	0	10/13/2023	7/19/2024	G2P1	20	12984	53	34	3	2	0	15	8	8	No	Yes	None	No	No	No	No		Cesarean Section	Placenta Previa	Negative	53	IVH 3	M	3.02	28
13	Patient_13	35	1/13/2024	1/15/2024	No	0	10/28/2023	8/3/2024	G2P1	15	12077	57	31	7	1	1	13	5	6	No	Yes	Pneumonia	Yes	No	No	No		Cesarean Section	Fetal Distress	Negative	110	MAS	F	2.54	38
14	Patient_14	35	1/14/2024	1/17/2024	Yes	37	11/12/2023	8/18/2024	G4P3	22	11235	51	33	4	2	1	3	2	7	No	Yes	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Fetal Distress	Negative	47	MAS	M	3.8	40
15	Patient_15	34	1/15/2024	1/17/2024	No	0	11/27/2023	9/2/2024	G2P1	18	11388	52	32	3	2	1	15	3	5	No	Yes	Septicemia	Yes	No	No	No		Induced with Oxytocin		Negative	75	HMD	F	3.16	32
16	Patient_16	35	1/16/2024	1/24/2024	Yes	53	12/12/2023	9/17/2024	G4P3	20	15642	60	21	6	1	0	1	5	8	Yes	No	None	No	No	No	No		Cesarean Section	Fetal Distress	Negative	56	NORMAL	F	4.45	40
17	Patient_17	27	1/17/2024	1/19/2024	No	0	12/27/2023	10/2/2024	G2P1	18	14037	50	32	7	2	0	15	4	7	No	Yes	None	Yes	No	No	No		Cesarean Section	Fetal Distress	Negative	122	IVH 1 WITH HMD	F	3.74	31
18	Patient_18	20	1/18/2024	1/21/2024	No	0	1/11/2024	10/17/2024	G2P1	23	14805	52	35	5	1	0	11	9	8	No	Yes	None	No	No	No	No		Induced with Oxytocin		Negative	70	MAS	M	2.5	41
19	Patient_19	21	1/19/2024	1/21/2024	No	0	1/26/2024	11/1/2024	G2P1	23	11762	67	28	2	1	0	14	9	8	No	Yes	Umbilical Sepsis	No	No	No	No		Spontaneous		Negative	120	HMD	F	2.87	30
20	Patient_20	35	1/20/2024	1/23/2024	No	0	2/10/2024	11/16/2024	G3P2	15	12848	50	24	6	2	1	17	8	8	No	Yes	Septicemia	No	No	No	No		Induced with Oxytocin		Negative	46	HMD WITH RDS	F	4.24	28
21	Patient_21	26	1/21/2024	1/26/2024	Yes	87	2/25/2024	12/1/2024	G1P0	20	10692	55	37	6	1	0	9	8	6	No	No	None	No	No	No	No		Cesarean Section	Breech	Negative	47	Normal	M	3.8	39
22	Patient_22	35	1/22/2024	1/29/2024	Yes	48	3/11/2024	12/16/2024	G1P0	23	21587	50	37	4	2	1	13	9	5	No	No	None	No	No	No	No		Induced with Oxytocin		Negative	91	NORMAL	F	3.29	38
23	Patient_23	35	1/23/2024	2/3/2024	Yes	81	3/26/2024	12/31/2024	G3P2	14	15719	56	34	2	1	1	8	1	7	No	No	Umbilical Sepsis	Yes	No	No	No		Induced with Oxytocin		Streptococcus	47	NORMAL	M	3.82	34
24	Patient_24	23	1/24/2024	1/30/2024	No	0	4/10/2024	1/15/2025	G1P0	18	28216	59	23	7	1	0	2	8	6	No	No	None	No	No	No	No		Cesarean Section	Breech	Negative	123	NORMAL	F	3.85	40
25	Patient_25	31	1/25/2024	2/4/2024	No	0	4/25/2024	1/30/2025	G2P1	17	13814	54	39	4	1	0	2	5	6	No	No	Septicemia	Yes	No	No	No		Induced with Oxytocin		Negative	80	NORMAL	M	2.32	32
26	Patient_26	26	1/26/2024	1/29/2024	No	0	5/10/2024	2/14/2025	G4P3	17	14374	69	29	8	2	1	1	3	5	No	Yes	Conjunctivitis	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	139	RDS	F	4.16	33
27	Patient_27	20	1/27/2024	1/29/2024	Yes	44	5/25/2024	3/1/2025	G4P3	23	13966	64	35	5	1	0	11	9	8	No	Yes	Umbilical Sepsis	No	No	No	No		Cesarean Section	Fetal Distress	Negative	132	TTN	M	2.05	35
28	Patient_28	34	1/28/2024	1/31/2024	Yes	33	6/9/2024	3/16/2025	G3P2	17	14278	65	23	4	2	1	16	9	8	No	Yes	Pneumonia	No	No	No	No		Spontaneous		Negative	96	RDS WITH HMD	F	4.36	28
29	Patient_29	28	1/29/2024	3/2/2024	Yes	83	6/24/2024	3/31/2025	G1P0	16	14596	64	35	3	1	0	6.9	9	5	No	No	Conjunctivitis	No	No	No	No		Spontaneous		Negative	129	NORMAL	F	4	32
30	Patient_30	28	1/30/2024	2/2/2024	Yes	27	7/9/2024	4/15/2025	G2P1	20	12252	65	23	7	1	1	3	8	8	No	Yes	Conjunctivitis	No	No	No	No		Cesarean Section	Placenta Previa	Negative	121	TTN	M	4.24	35
31	Patient_31	35	1/31/2024	7/2/2024	Yes	21	7/24/2024	4/30/2025	G1P0	14	22763	60	40	4	1	1	14.5	8	9	No	No	Septicemia	No	No	No	No		Induced with Oxytocin		Negative	119	Normal	M	2.06	38
32	Patient_32	23	1/2/2024	1/13/2024	No	0	8/8/2024	5/15/2025	G1P0	15	14981	52	27	5	1	0	4	4	5	No	No	Pneumonia	Yes	No	No	No		Cesarean Section	Breech	Acinetobacter	139	IVH 1	F	2.08	30
33	Patient_33	32	2/2/2024	5/2/2024	Yes	66	8/23/2024	5/30/2025	G2P1	17	28923	51	20	8	2	0	11.9	5	9	No	No	Conjunctivitis	No	No	No	No		Spontaneous		Negative	73	Normal	M	2.02	40
34	Patient_34	25	3/2/2024	3/12/2024	No	0	9/7/2024	6/14/2025	G1P0	14	15673	51	32	5	2	0	16	0	7	Yes	No	None	Yes	No	No	No		Spontaneous		Negative	55	Normal	F	2.23	30
35	Patient_35	31	4/2/2024	12/2/2024	No	0	9/22/2024	6/29/2025	G1P0	22	17678	62	38	4	2	0	12.7	9	9	No	No	Septicemia	No	No	No	No		Spontaneous		Negative	119	Normal	M	2.25	38
36	Patient_36	20	5/2/2024	5/16/2024	Yes	89	10/7/2024	7/14/2025	G1P0	23	13122	52	31	7	2	0	9.6	2	7	No	No	Septicemia	Yes	No	No	No		Spontaneous		Streptococcus	87	Normal	M	4.15	30
37	Patient_37	35	6/2/2024	6/4/2024	No	0	10/22/2024	7/29/2025	G2P1	16	11434	64	20	6	2	1	11	9	8	No	Yes	Pneumonia	No	No	No	No		Cesarean Section	Breech	Negative	66	MAS	F	3.45	40
38	Patient_38	32	7/2/2024	7/5/2024	Yes	80	11/6/2024	8/13/2025	G1P0	20	13150	70	23	5	2	1	8.5	9	8	No	Yes	Umbilical Sepsis	No	Yes	No	No		Induced with Oxytocin		Negative	118	IVH 3 WITH HMD	F	3.57	28
39	Patient_39	35	8/2/2024	9/2/2024	No	0	11/21/2024	8/28/2025	G3P2	19	21129	55	24	6	1	1	10.6	3	5	No	No	None	No	No	No	No		Induced with Oxytocin		Negative	70	RDS	F	3.32	30
40	Patient_40	20	9/2/2024	11/2/2024	Yes	34	12/1/2024	9/7/2025	G1P0	16	22545	55	38	8	1	1	14.7	9	6	No	No	Conjunctivitis	No	No	No	No		Spontaneous		Negative	109	Normal	F	3.33	31
41	Patient_41	35	10/2/2024	12/2/2024	No	0	12/1/2024	9/7/2025	G1P0	16	24879	58	39	5	2	1	13.1	0	10	No	YES	Umbilical Sepsis	No	No	No	No		Cesarean Section	Breech	Negative	130	IVH 2 WITH HMD	F	2.78	28
42	Patient_42	34	11/2/2024	2/16/2024	No	0	12/1/2024	9/7/2025	G1P0	21	16836	54	27	4	2	0	18	0	9	No	No	Conjunctivitis	No	No	No	No		Cesarean Section	Breech	Negative	102	Abnormal	F	2.12	37
43	Patient_43	35	12/2/2024	12/4/2024	No	0	12/1/2024	9/7/2025	G4P3	14	15859	51	28	3	2	1	18	4	6	No	Yes	Conjunctivitis	Yes	No	No	No		Spontaneous		Negative	125	IVH 3 WITH HMD	F	3.25	28
44	Patient_44	20	2/13/2024	2/16/2024	Yes	58	12/1/2024	9/7/2025	G4P3	22	13785	64	34	8	1	1	4	7	8	No	Yes	None	No	No	No	No		Spontaneous		Negative	134	MAS	F	3.8	40
45	Patient_45	24	2/14/2024	2/16/2024	No	0	12/1/2024	9/7/2025	G1P0	23	13853	57	34	6	2	1	16	9	8	No	Yes	Pneumonia	No	No	No	No		Cesarean Section	Placenta Previa	Negative	66	TTN	F	2.17	36
46	Patient_46	35	2/15/2024	2/18/2024	Yes	60	12/1/2024	9/7/2025	G4P3	20	12329	60	28	7	1	1	3	7	8	No	Yes	Septicemia	No	No	No	No		Induced with Oxytocin		Negative	109	NORMAL	F	2.5	35
47	Patient_47	34	2/16/2024	2/29/2024	Yes	60	12/1/2024	9/7/2025	G2P1	16	12358	52	33	6	2	0	11.7	2	7	No	No	Umbilical Sepsis	Yes	No	No	No		Spontaneous		Klebsiella	102	NORMAL	F	2.85	34
48	Patient_48	32	2/17/2024	2/21/2024	Yes	46	12/1/2024	9/7/2025	G2P1	20	23288	66	33	7	1	0	4	8	6	No	No	None	No	No	No	No		Cesarean Section	Breech	Negative	98	Normal	F	3.23	33
49	Patient_49	33	2/18/2024	2/20/2024	Yes	31	12/1/2024	9/7/2025	G3P2	21	13906	58	39	7	2	1	3	7	8	No	Yes	Umbilical Sepsis	No	No	No	No		Cesarean Section	Breech	Negative	44	TTN	F	3.08	39
50	Patient_50	30	2/19/2024	3/1/2024	No	0	12/1/2024	9/7/2025	G2P1	17	13482	64	35	8	2	1	13	3	7	No	No	Septicemia	Yes	No	No	No		Induced with Oxytocin		Streptococcus	89	Normal	M	2.62	41
51	Patient_51	18	2/20/2024	3/5/2024	Yes	36	12/1/2024	9/7/2025	G1P0	18	15542	65	39	4	2	0	10.7	1	7	No	No	Septicemia	Yes	No	No	No		Induced with Oxytocin		Klebsiella	91	Normal	M	2.44	36
52	Patient_52	32	2/21/2024	3/2/2024	No	0	12/1/2024	9/7/2025	G3P2	17	13017	51	20	8	1	1	20	5	6	No	No	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	116	NORMAL	M	3.86	38
53	Patient_53	31	2/22/2024	3/4/2024	Yes	40	12/1/2024	9/7/2025	G2P1	21	12331	58	25	3	1	0	16	2	5	No	No	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Fetal Distress	Streptococcus	70	IVH 2	M	3.3	28
54	Patient_54	29	2/23/2024	2/26/2024	No	0	12/1/2024	9/7/2025	G1P0	15	15344	58	20	3	1	1	4	5	5	No	Yes	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Negative	107	RDS	F	3.49	29
55	Patient_55	18	2/24/2024	3/7/2024	Yes	39	12/1/2024	9/7/2025	G1P0	21	15261	51	35	4	2	0	2	5	7	No	No	Conjunctivitis	Yes	No	No	No		Induced with Oxytocin		Streptococcus	89	NORMAL	M	2.5	40
56	Patient_56	34	2/25/2024	2/28/2024	No	0	12/1/2024	9/7/2025	G2P1	19	26416	53	34	6	2	1	5	6	10	No	No	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	48	RDS	F	4.04	30
57	Patient_57	35	2/26/2024	2/28/2024	No	0	12/1/2024	9/7/2025	G1P0	18	12358	64	34	7	2	0	14	1	7	No	Yes	Conjunctivitis	Yes	No	No	No		Cesarean Section	Breech	Negative	43	RDS	F	2.53	31
58	Patient_58	35	2/27/2024	3/1/2024	No	0	12/1/2024	9/7/2025	G4P3	21	12019	62	39	2	2	1	5	8	8	No	Yes	Umbilical Sepsis	No	No	No	No		Spontaneous		Negative	111	TTN	F	2.42	39
59	Patient_59	23	2/28/2024	3/12/2024	Yes	83	12/1/2024	9/7/2025	G4P3	18	15637	66	35	2	2	1	9.2	0	7	No	No	None	Yes	No	No	No		Induced with Oxytocin		E. coli	68	Normal	F	3.08	40
60	Patient_60	28	2/29/2024	1/3/2024	Yes	89	12/1/2024	9/7/2025	G2P1	18	19784	58	25	2	1	0	8.5	2	8	No	No	Pneumonia	No	No	No	No		Cesarean Section	Placenta Previa	Negative	131	Abnormal	M	3.28	34
61	Patient_61	34	1/3/2024	1/5/2024	No	0	12/1/2024	9/7/2025	G4P3	24	11758	56	30	3	2	1	14	7	8	No	Yes	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	108	IVH 2 WITH RDS	M	3.03	28
62	Patient_62	29	2/3/2024	2/6/2024	No	0	12/1/2024	9/7/2025	G3P2	17	14177	60	30	5	2	0	15	9	8	No	Yes	Septicemia	No	Yes	No	No		Spontaneous		Negative	92	IVH WITH HMD	F	2.62	29
63	Patient_63	22	3/3/2024	3/5/2024	Yes	38	12/1/2024	9/7/2025	G3P2	24	13128	67	27	8	1	0	2	4	7	No	Yes	Conjunctivitis	Yes	No	No	No		Induced with Oxytocin		Negative	121	RDS	F	3.43	30
64	Patient_64	28	4/3/2024	4/6/2024	Yes	89	12/1/2024	9/7/2025	G4P3	24	13180	50	25	8	1	1	8.1	7	8	No	Yes	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	43	RDS	F	2.35	30
65	Patient_65	35	5/3/2024	5/14/2024	No	0	12/1/2024	9/7/2025	G4P3	15	15892	69	40	3	2	1	4	3	6	No	No	Umbilical Sepsis	Yes	Yes	No	No		Spontaneous		Acinetobacter	72	IVH 2 WITH HMD	F	3.83	29
66	Patient_66	24	6/3/2024	6/6/2024	Yes	89	12/1/2024	9/7/2025	G3P2	16	11137	56	27	2	2	1	9.6	1	5	No	Yes	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Breech	Negative	83	IVH 1 WITH TTN	F	3.38	35
67	Patient_67	27	7/3/2024	7/5/2024	No	0	12/1/2024	9/7/2025	G2P1	15	14472	63	40	3	1	1	18	4	7	No	Yes	None	Yes	Yes	No	No		Spontaneous		Negative	52	IVH 1 WITH RDS	M	4.13	28
68	Patient_68	28	8/3/2024	8/6/2024	Yes	53	12/1/2024	9/7/2025	G2P1	15	13832	57	33	8	1	1	14	1	7	No	Yes	Pneumonia	Yes	No	No	No		Spontaneous		Negative	105	TTN	F	3.75	35
69	Patient_69	35	9/3/2024	9/5/2024	Yes	27	12/1/2024	9/7/2025	G3P2	22	14579	50	32	3	1	1	2	1	7	No	Yes	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Breech	Negative	64	Normal	M	2.13	37
70	Patient_70	32	10/3/2024	3/15/2024	Yes	21	12/1/2024	9/7/2025	G3P2	17	10607	68	28	6	1	1	12	8	8	No	No	Conjunctivitis	No	No	No	No		Cesarean Section	Fetal Distress	Negative	72	RDS	M	2.41	30
71	Patient_71	19	11/3/2024	11/5/2024	No	0	12/1/2024	9/7/2025	G3P2	24	14031	64	24	6	1	1	17	3	5	No	Yes	None	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	86	Normal	M	2.52	33
72	Patient_72	20	12/3/2024	12/6/2024	No	0	12/1/2024	9/7/2025	G4P3	24	15164	58	23	2	2	1	15	4	6	No	Yes	Septicemia	Yes	No	No	No		Cesarean Section	Fetal Distress	Negative	107	Normal	M	3.5	34
73	Patient_73	22	3/13/2024	3/15/2024	Yes	72	12/1/2024	9/7/2025	G3P2	23	22141	54	36	2	2	1	1	3	7	No	No	Pneumonia	No	No	No	No		Spontaneous		Negative	126	Normal	F	4.45	34
74	Patient_74	35	3/14/2024	3/17/2024	No	0	12/1/2024	9/7/2025	G1P0	18	13593	62	40	8	1	1	19	8	8	No	Yes	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	80	MAS	M	2.33	41
75	Patient_75	35	3/15/2024	3/29/2024	No	0	12/1/2024	9/7/2025	G3P2	23	15300	50	32	3	2	0	13.9	0	6	No	No	Umbilical Sepsis	Yes	No	No	No		Spontaneous		Acinetobacter	102	TTN	F	3.55	35
76	Patient_76	23	3/16/2024	3/17/2024	Yes	37	12/1/2024	9/7/2025	G1P0	21	19386	70	22	6	1	1	16	0	9	No	No	None	No	No	No	No		Induced with Oxytocin		Negative	74	Normal	F	3.31	37
77	Patient_77	30	3/17/2024	3/24/2024	Yes	53	12/1/2024	9/7/2025	G1P0	15	13474	59	40	2	2	1	11	6	8	No	No	Conjunctivitis	No	Yes	No	No		Spontaneous		Negative	53	RDS	M	3.49	29
78	Patient_78	19	3/18/2024	3/21/2024	Yes	19	12/1/2024	9/7/2025	G1P0	21	14818	65	22	8	2	1	20	4	7	No	Yes	Conjunctivitis	Yes	No	No	No		Induced with Oxytocin		Negative	109	Normal	F	3.69	36
79	Patient_79	35	3/19/2024	3/21/2024	No	0	12/1/2024	9/7/2025	G1P0	21	15138	60	33	3	1	1	15	3	6	No	Yes	Pneumonia	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	74	HMD	F	2.78	28
80	Patient_80	31	3/20/2024	3/31/2024	No	0	12/1/2024	9/7/2025	G3P2	21	14823	69	24	6	2	0	2	5	5	No	No	Pneumonia	Yes	Yes	No	No		Cesarean Section	Breech	Streptococcus	46	RDS	F	2.28	29
81	Patient_81	23	3/21/2024	3/23/2024	No	0	12/1/2024	9/7/2025	G1P0	20	12906	54	28	2	2	1	12	8	8	No	Yes	Septicemia	No	No	No	No		Induced with Oxytocin		Negative	100	HMD	F	3.83	28
82	Patient_82	20	3/22/2024	3/25/2024	Yes	65	12/1/2024	9/7/2025	G2P1	23	11760	62	20	8	2	1	1	5	7	No	Yes	None	Yes	No	No	No		Spontaneous		Negative	53	RDS	M	2.22	31
83	Patient_83	35	3/23/2024	3/25/2024	No	0	12/1/2024	9/7/2025	G1P0	14	14445	61	26	8	2	1	5	5	7	No	Yes	Septicemia	Yes	No	No	No		Spontaneous		Negative	40	Normal	M	2.89	33
84	Patient_84	22	3/24/2024	4/7/2024	No	0	12/1/2024	9/7/2025	G1P0	16	11899	50	39	8	2	0	10.8	0	6	No	No	Septicemia	Yes	No	No	No		Induced with Oxytocin		Streptococcus	81	Normal	M	4.45	40
85	Patient_85	29	3/25/2024	3/27/2024	No	0	12/1/2024	9/7/2025	G3P2	16	11359	51	32	2	2	0	19	9	8	No	Yes	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	66	TTN	M	3.08	38
86	Patient_86	21	3/26/2024	3/31/2024	Yes	18	12/1/2024	9/7/2025	G1P0	18	15448	70	20	6	2	0	11	1	10	No	No	None	No	No	No	No		Induced with Oxytocin		Negative	79	TTN	F	2.44	38
87	Patient_87	26	3/27/2024	3/29/2024	Yes	89	12/1/2024	9/7/2025	G1P0	22	13947	64	38	2	2	0	7.2	0	6	No	Yes	Septicemia	Yes	No	No	No		Spontaneous		Negative	86	MAS	M	4.12	41
88	Patient_88	25	3/28/2024	3/31/2024	Yes	20	12/1/2024	9/7/2025	G1P0	17	12719	51	40	3	1	1	16	0	6	No	Yes	Umbilical Sepsis	Yes	No	No	No		Spontaneous		Negative	53	RDS	F	4.23	30
89	Patient_89	24	3/29/2024	4/11/2024	Yes	22	12/1/2024	9/7/2025	G2P1	14	11280	69	27	4	1	1	11.2	4	5	No	No	Septicemia	Yes	No	No	No		Induced with Oxytocin		Streptococcus	52	TTN	F	2.16	39
90	Patient_90	35	3/30/2024	4/2/2024	Yes	89	12/1/2024	9/7/2025	G3P2	16	14266	65	36	5	1	0	9	0	6	No	Yes	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	94	TTN	F	3.29	37
91	Patient_91	34	3/31/2024	4/2/2024	Yes	85	12/1/2024	9/7/2025	G3P2	15	12887	56	32	2	2	0	6.1	5	7	No	Yes	Pneumonia	Yes	No	No	No		Cesarean Section	Fetal Distress	Negative	65	Normal	F	3.17	40
92	Patient_92	35	1/4/2024	1/7/2024	No	0	12/1/2024	9/7/2025	G1P0	22	14266	51	37	5	2	0	2	8	8	No	Yes	Pneumonia	No	No	No	No		Cesarean Section	Placenta Previa	Negative	53	Normal	M	4	33
93	Patient_93	29	2/4/2024	2/16/2024	Yes	83	12/1/2024	9/7/2025	G1P0	15	11666	59	33	7	1	0	8.3	3	6	No	No	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Breech	Klebsiella	73	Normal	F	3.56	31
94	Patient_94	24	3/4/2024	3/7/2024	No	0	12/1/2024	9/7/2025	G2P1	14

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125	Patient_125	33	4/5/2024	11/5/2024	No	0	12/1/2024	9/7/2025	G4P3	14	26425	61	28	6	2	1	13.6	4	9	No	No	Pneumonia	No	No	No	No		Spontaneous		Negative	49	Normal	M	4.32	37
126	Patient_126	25	5/5/2024	5/17/2024	Yes	89	12/1/2024	9/7/2025	G4P3	14	15432	65	24	6	2	1	8.9	1	6	No	No	None	Yes	No	No	No		Spontaneous		Klebsiella	69	Normal	M	2.58	34
127	Patient_127	20	6/5/2024	12/5/2024	Yes	22	12/1/2024	9/7/2025	G4P3	24	17580	70	26	3	1	0	13.2	5	7	No	No	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	105	TTN	F	3.5	39
128	Patient_128	22	7/5/2024	7/8/2024	Yes	73	12/1/2024	9/7/2025	G1P0	16	13055	55	25	2	1	1	8.8	1	6	No	Yes	Umbilical Sepsis	Yes	No	No	No		Induced with Oxytocin		Negative	41	RDS	F	3.09	32
129	Patient_129	18	8/5/2024	8/7/2024	No	0	12/1/2024	9/7/2025	G2P1	23	11624	70	35	2	1	0	16	9	8	No	Yes	Conjunctivitis	No	No	No	No		Spontaneous		Negative	98	TTN	M	3.25	34
130	Patient_130	28	9/5/2024	9/15/2024	No	0	12/1/2024	9/7/2025	G3P2	19	12276	50	22	8	2	1	2	0	7	No	No	Conjunctivitis	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	93	RDS	F	3.64	32
131	Patient_131	18	10/5/2024	10/7/2024	Yes	23	12/1/2024	9/7/2025	G1P0	18	12715	57	32	7	2	1	3	7	8	No	Yes	Conjunctivitis	No	No	No	No		Spontaneous		Negative	122	Normal	F	2.34	35
132	Patient_132	31	11/5/2024	11/8/2024	Yes	89	12/1/2024	9/7/2025	G4P3	18	13766	56	30	6	2	0	8.2	9	8	No	Yes	Conjunctivitis	No	Yes	No	No		Cesarean Section	Fetal Distress	Negative	134	Normal	F	2.34	28
133	Patient_133	30	12/5/2024	12/7/2024	No	0	12/1/2024	9/7/2025	G1P0	22	11302	65	29	7	1	1	4	1	5	No	Yes	None	Yes	No	No	No		Induced with Oxytocin		Negative	123	MAS	F	2.4	41
134	Patient_134	35	5/13/2024	5/15/2024	No	0	12/1/2024	9/7/2025	G1P0	21	18403	69	39	8	2	0	4	6	8	No	No	Pneumonia	No	No	No	No		Cesarean Section	Breech	Negative	56	RDS	M	4.38	33
135	Patient_135	25	5/14/2024	5/15/2024	No	0	12/1/2024	9/7/2025	G2P1	24	28301	62	39	2	1	1	17	8	8	No	No	Conjunctivitis	No	No	No	No		Cesarean Section	Breech	Negative	96	Normal	M	3.5	34
136	Patient_136	27	5/15/2024	5/24/2024	No	0	12/1/2024	9/7/2025	G1P0	19	25359	66	36	7	1	0	16	4	5	No	No	Septicemia	No	No	No	No		Spontaneous		Negative	100	Normal	F	4.02	35
137	Patient_137	31	5/16/2024	5/29/2024	Yes	21	12/1/2024	9/7/2025	G2P1	22	11638	69	37	6	2	0	11.3	4	7	No	No	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Streptococcus	106	TTN	F	2.3	38
138	Patient_138	27	5/17/2024	5/23/2024	Yes	77	12/1/2024	9/7/2025	G2P1	15	21663	53	24	6	1	1	7.4	9	6	No	No	Conjunctivitis	No	No	No	No		Cesarean Section	Breech	Negative	129	RDS	M	3.95	33
139	Patient_139	27	5/18/2024	5/20/2024	Yes	88	12/1/2024	9/7/2025	G3P2	24	11055	67	20	8	1	0	6.7	2	5	No	Yes	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Negative	90	Normal	M	3.18	40
140	Patient_140	20	5/19/2024	5/30/2024	Yes	72	12/1/2024	9/7/2025	G3P2	15	15821	50	40	5	2	0	2	2	7	No	No	Conjunctivitis	Yes	No	No	No		Cesarean Section	Fetal Distress	Streptococcus	128	HMD	M	2.71	30
141	Patient_141	35	5/20/2024	5/22/2024	No	0	12/1/2024	9/7/2025	G2P1	21	11525	64	39	3	1	1	16	2	6	No	Yes	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Breech	Negative	124	Normal	M	2.48	38
142	Patient_142	35	5/21/2024	5/31/2024	No	0	12/1/2024	9/7/2025	G4P3	17	15569	66	20	4	2	0	5	1	7	No	No	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Negative	63	Normal	F	4.28	34
143	Patient_143	24	5/22/2024	5/24/2024	Yes	32	12/1/2024	9/7/2025	G2P1	24	11027	61	30	7	2	1	16	9	8	No	Yes	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	121	Normal	F	4.12	30
144	Patient_144	35	5/23/2024	6/4/2024	No	0	12/1/2024	9/7/2025	G4P3	20	13125	53	29	2	1	0	5	5	6	No	No	Septicemia	Yes	No	No	No		Spontaneous		E. coli	109	HMD	M	4.49	28
145	Patient_145	24	5/24/2024	5/26/2024	Yes	46	12/1/2024	9/7/2025	G3P2	16	14287	58	25	3	2	1	1	7	8	No	Yes	Conjunctivitis	No	No	No	No		Cesarean Section	Placenta Previa	Negative	45	Normal	F	4.16	41
146	Patient_146	27	5/25/2024	5/31/2024	Yes	89	12/1/2024	9/7/2025	G1P0	19	20747	60	29	7	2	0	9.6	1	5	No	No	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	85	Normal	F	3.74	31
147	Patient_147	35	5/26/2024	6/7/2024	Yes	89	12/1/2024	9/7/2025	G4P3	21	11364	60	32	3	2	0	8.3	0	6	No	No	None	Yes	No	No	No		Spontaneous		Klebsiella	101	TTN	F	3.58	35
148	Patient_148	35	5/27/2024	4/6/2024	Yes	64	12/1/2024	9/7/2025	G3P2	14	27542	50	26	5	2	0	2	6	10	No	No	Septicemia	No	No	No	No		Spontaneous		Negative	112	Normal	M	4.03	39
149	Patient_149	31	5/28/2024	6/10/2024	Yes	65	12/1/2024	9/7/2025	G1P0	20	15621	53	37	8	2	1	12	0	5	No	No	Conjunctivitis	Yes	No	No	Yes	Prematurity	Cesarean Section	Placenta Previa	Streptococcus	60	Normal	M	2.93	34
150	Patient_150	35	5/29/2024	6/1/2024	No	0	12/1/2024	9/7/2025	G3P2	19	13746	58	25	8	1	1	2	9	8	No	Yes	Septicemia	No	No	No	No		Spontaneous		Negative	54	Normal	M	2.22	32
151	Patient_151	19	5/30/2024	6/1/2024	No	0	12/1/2024	9/7/2025	G3P2	18	15978	61	26	2	1	1	17	3	7	No	Yes	Conjunctivitis	Yes	No	No	No		Spontaneous		Negative	124	Normal	M	3.96	35
152	Patient_152	18	5/31/2024	6/11/2024	No	0	12/1/2024	9/7/2025	G2P1	22	12646	60	28	4	1	1	16	2	7	No	No	Pneumonia	Yes	Yes	No	No		Spontaneous		Streptococcus	131	HMD	M	3.75	28
153	Patient_153	35	1/6/2024	1/18/2024	No	0	12/1/2024	9/7/2025	G3P2	17	11918	53	31	8	1	0	17	0	6	No	No	Pneumonia	Yes	No	No	No		Cesarean Section	Breech	Streptococcus	131	HMD	M	3.62	29
154	Patient_154	35	2/6/2024	2/9/2024	Yes	45	12/1/2024	9/7/2025	G3P2	16	13797	53	35	7	1	0	3	9	8	No	Yes	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	40	HMD	F	4.11	29
155	Patient_155	34	3/6/2024	3/19/2024	No	0	12/1/2024	9/7/2025	G3P2	17	12531	62	29	3	1	0	12.3	4	7	No	No	Conjunctivitis	Yes	Yes	No	No		Induced with Oxytocin		Klebsiella	49	HMD	F	4.34	29
156	Patient_156	28	4/6/2024	6/6/2024	No	0	12/1/2024	9/7/2025	G4P3	22	19410	62	21	3	2	0	14.7	1	7	No	No	None	No	No	No	No		Spontaneous		Negative	60	NORMAL	F	4.11	39
157	Patient_157	32	5/6/2024	5/8/2024	No	0	12/1/2024	9/7/2025	G3P2	16	14345	53	27	8	1	1	4	8	8	No	Yes	None	No	No	No	No		Spontaneous		Negative	100	Normal	F	2.71	37
158	Patient_158	28	6/6/2024	6/9/2024	No	0	12/1/2024	9/7/2025	G1P0	15	14577	61	36	3	1	1	3	7	8	No	Yes	Umbilical Sepsis	No	No	No	No		Cesarean Section	Fetal Distress	Negative	105	Normal	F	4.13	41
159	Patient_159	35	7/6/2024	8/6/2024	No	0	12/1/2024	9/7/2025	G4P3	17	25950	52	27	4	2	0	10.8	0	9	No	No	Umbilical Sepsis	No	No	No	No		Spontaneous		Negative	131	RDS	F	3.85	31
160	Patient_160	35	8/6/2024	8/18/2024	Yes	65	12/1/2024	9/7/2025	G2P1	14	12906	62	24	2	1	0	16	1	6	No	No	None	Yes	No	No	No		Spontaneous		Streptococcus	113	Normal	M	3.18	33
161	Patient_161	26	9/6/2024	11/6/2024	Yes	37	12/1/2024	9/7/2025	G4P3	21	24928	57	34	8	1	0	12.9	7	8	No	No	Umbilical Sepsis	No	Yes	No	No		Spontaneous		Negative	97	HMD	M	4.25	29
162	Patient_162	35	10/6/2024	10/18/2024	No	0	12/1/2024	9/7/2025	G1P0	17	14086	54	22	7	2	1	13	4	5	No	No	Conjunctivitis	Yes	No	No	No		Spontaneous		Streptococcus	59	NORMAL	F	3.41	40
163	Patient_163	25	11/6/2024	11/8/2024	Yes	89	12/1/2024	9/7/2025	G3P2	24	15580	66	24	6	1	1	8	0	5	No	Yes	Pneumonia	Yes	No	No	No		Spontaneous		Negative	123	Normal	M	2.02	40
164	Patient_164	35	12/6/2024	12/19/2024	No	0	12/1/2024	9/7/2025	G3P2	21	13075	66	25	5	2	1	13.1	0	5	No	No	Septicemia	Yes	No	No	No		Cesarean Section	Breech	Streptococcus	45	RDS	M	4.32	31
165	Patient_165	35	6/13/2024	6/27/2024	No	0	12/1/2024	9/7/2025	G2P1	18	11713	69	38	3	2	0	10.1	4	7	No	No	Umbilical Sepsis	Yes	Yes	No	No		Spontaneous		Streptococcus	137	HMD	F	2.05	28
166	Patient_166	35	6/14/2024	6/26/2024	No	0	12/1/2024	9/7/2025	G1P0	23	12062	57	25	2	1	0	4	1	5	No	No	Conjunctivitis	Yes	No	No	No		Spontaneous		Klebsiella	92	NORMAL	F	2.92	37
167	Patient_167	35	6/15/2024	6/17/2024	No	0	12/1/2024	9/7/2025	G3P2	15	12976	57	20	5	1	1	19	9	8	No	Yes	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	81	NORMAL	F	3.63	41
168	Patient_168	35	6/16/2024	6/19/2024	No	0	12/1/2024	9/7/2025	G2P1	15	12296	54	39	8	1	0	4	9	8	No	Yes	Septicemia	No	No	No	No		Induced with Oxytocin		Negative	61	NORMAL	F	3.66	34
169	Patient_169	35	6/17/2024	6/18/2024	Yes	64	12/1/2024	9/7/2025	G1P0	24	14821	54	27	4	1	0	11	9	9	No	No	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	53	Normal	F	3	41
170	Patient_170	35	6/18/2024	6/23/2024	No	0	12/1/2024	9/7/2025	G1P0	18	25074	54	28	4	2	1	17	4	6	No	No	Conjunctivitis	No	No	No	No		Cesarean Section	Placenta Previa	Negative	86	NORMAL	F	3.93	40
171	Patient_171	24	6/19/2024	6/24/2024	No	0	12/1/2024	9/7/2025	G3P2	18	23382	54	21	2	2	1	1	4	7	No	No	Conjunctivitis	No	No	No	No		Cesarean Section	Placenta Previa	Negative	113	Normal	F	2.64	35
172	Patient_172	24	6/20/2024	6/30/2024	No	0	12/1/2024	9/7/2025	G4P3	20	11125	68	27	7	2	1	13	2	6	No	No	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Negative	106	Normal	F	2.25	30
173	Patient_173	26	6/21/2024	6/25/2024	Yes	39	12/1/2024	9/7/2025	G3P2	20	24501	51	31	8	1	0	1	7	5	No	No	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	138	Normal	M	2.46	36
174	Patient_174	20	6/22/2024	7/6/2024	Yes	69	12/1/2024	9/7/2025	G4P3	19	15632	63	39	2	2	0	14.4	4	7	No	No	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Fetal Distress	Streptococcus	136	RDS	M	2.29	31
175	Patient_175	35	6/23/2024	2/7/2024	No	0	12/1/2024	9/7/2025	G2P1	22	22816	54	29	6	1	0	17	6	10	No	No	Septicemia	No	No	No	No		Cesarean Section	Fetal Distress	Negative	106	HMD	F	3.76	28
176	Patient_176	35	6/24/2024	6/27/2024	No	0	12/1/2024	9/7/2025	G3P2	14	14596	53	21	6	1	0	3	5	6	No	Yes	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Negative	136	NORMAL	F	3.22	40
177	Patient_177	35	6/25/2024	6/27/2024	No	0	12/1/2024	9/7/2025	G1P0	18	14283	54	30	8	1	0	20	1	7	No	Yes	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	125	NORMAL	F	3.87	35
178	Patient_178	20	6/26/2024	7/8/2024	Yes	73	12/1/2024	9/7/2025	G3P2	19	12603	68	33	5	1	1	9.7	4	7	No	No	Septicemia	Yes	No	No	No		Cesarean Section	Fetal Distress	E. coli	54	MAS	M	2.3	41
179	Patient_179	35	6/27/2024	7/8/2024	No	0	12/1/2024	9/7/2025	G3P2	24	11066	59	39	6	2	0	3	2	5	No	No	Pneumonia	Yes	No	No	No		Cesarean Section	Fetal Distress	Streptococcus	60	Normal	F	3.9	34
180	Patient_180	18	6/28/2024	7/1/2024	No	0	12/1/2024	9/7/2025	G1P0	22	11232	51	28	5	1	1	2	4	5	No	Yes	None	Yes	No	No	No		Spontaneous		Negative	50	Normal	F	2.71	40
181	Patient_181	30	6/29/2024	7/1/2024	No	0	12/1/2024	9/7/2025	G3P2	14	13156	52	32	2	1	0	12	8	8	No	Yes	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	128	NORMAL	M	2.76	31
182	Patient_182	22	6/30/2024	7/13/2024	No	0	12/1/2024	9/7/2025	G2P1	18	12169	55	24	5	1	0	12.8	0	7	No	No	Pneumonia	Yes	No	No	No		Cesarean Section	Breech	Streptococcus	64	RDS	F	2.23	33
183	Patient_183	35	1/7/2024	1/21/2024	No	0	12/1/2024	9/7/2025	G1P0	18	13158	59	22	5	2	1	13.5	0	7	No	No	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Streptococcus	99	NORMAL	F	4.3	41
184	Patient_184	34	2/7/2024	2/10/2024	No	0	12/1/2024	9/7/2025	G2P1	17	13275	59	40	4	2	1	5	8	8	No	Yes	Conjunctivitis	No	No	No	No		Spontaneous		Negative	83	NORMAL	M	2.49	38
185	Patient_185	35	3/7/2024	5/7/2024	No	0	12/1/2024	9/7/2025	G1P0	15	21082	64	39	6	1	1	14.6	2	6	No	No	Pneumonia	No	No	No	No		Spontaneous		Negative	113	Normal	M	3.47	38
186	Patient_186	31	4/7/2024	12/7/2024	Yes	24	12/1/2024	9/7/2025	G2P1	18	20691	55	35	6	1	0	13.5	2	9	No	No	Septicemia	No	No	No	No		Spontaneous		Negative	130	Normal	F	2.39	37
187	Patient_187	18	5/7/2024	5/9/2024	Yes	52	12/1/2024	9/7/2025	G1P0	18	11110	61	39	8	2	0	11	9	8	No	Yes	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	114	Normal	F	2.11	34
188	Patient_188	19	6/7/2024	6/18/2024	No	0	12/1/2024	9/7/2025	G4P3	16	12493	53	24	2	1	1	12	4	5	No	No	Pneumonia	Yes	Yes	No	No		Induced with Oxytocin		Streptococcus	113	HMD	F	3.61	29
189	Patient_189	29	7/7/2024	7/9/2024	No	0	12/1/2024	9/7/2025	G1P0	21	13081	59	31	7	2	1	5	9	8	No	Yes	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	132	Normal	M	3.44	41
190	Patient_190	19	8/7/2024	8/17/2024	No	0	12/1/2024	9/7/2025	G4P3	15	15718	58	27	6	2	0	4	4	6	No	No	None	Yes	No	No	No		Spontaneous		Negative	117	Normal	F	2.17	35
191	Patient_191	18	9/7/2024	11/7/2024	No	0	12/1/2024	9/7/2025	G2P1	15	28820	59	22	8	1	0	10.8	4	10	No	No	Septicemia	No	No	No	No		Spontaneous		Negative	42	RDS	F	3.05	30
192	Patient_192	35	10/7/2024	7/16/2024	No	0	12/1/2024	9/7/2025	G3P2	21	16214	51	40	5	2	0	13	4	5	No	No	Umbilical Sepsis	No	No	No	No		Cesarean Section	Fetal Distress	Negative	54	Normal	F	2.71	37
193	Patient_193	35	11/7/2024	11/9/2024	Yes	75	12/1/2024	9/7/2025	G1P0	18	14847	56	23	2	2	1	8.3	8	8	No	Yes	Conjunctivitis	No	Yes	No	No		Induced with Oxytocin		Negative	68	HMD	M	3.21	28
194	Patient_194	35	12/7/2024	7/20/2024	No	0	12/1/2024	9/7/2025	G1P0	23	14546	69	37	5	1	0	20	2	9	No	No	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	47	Normal	F	2.58	36
195	Patient_195	35	7/13/2024	7/14/2024	No	0	12/1/2024	9/7/2025	G2P1	16	10802	69	39	2	1	0	11	8	6	No	No	Septicemia	No	No	No	No		Cesarean Section	Fetal Distress	Negative	88	NORMAL	F	3.95	33
196	Patient_196	32	7/14/2024	7/26/2024	Yes	61	12/1/2024	9/7/2025	G1P0	17	15007	66	33	3	1	1	17	0	6	No	No	Umbilical Sepsis	Yes	No	No	No		Spontaneous		E. coli	47	Normal	F	3.49	35
197	Patient_197	35	7/15/2024	7/28/2024	No	0	12/1/2024	9/7/2025	G3P2	14	13472	52	21	3	2	0	13	0	7	No	No	None	Yes	Yes	No	No		Spontaneous		Klebsiella	59	HMD	M	4.43	28
198	Patient_198	22	7/16/2024	7/18/2024	No	0	12/1/2024	9/7/2025	G2P1	14	24959	60	30	4	1	0	3	3	8	No	No	Septicemia	No	No	No	No		Cesarean Section	Breech	Negative	127	NORMAL	F	4.21	40
199	Patient_199	22	7/17/2024	7/19/2024	Yes	64	12/1/2024	9/7/2025	G1P0	20	13660	54	29	2	2	1	1	9	8	No	Yes	Pneumonia	No	No	No	No		Cesarean Section	Breech	Negative	77	RDS	F	2.24	30
200	Patient_200	35	7/18/2024	7/21/2024	No	0	12/1/2024	9/7/2025	G3P2	22	12541	54	22	6	2	0	3	8	8	No	Yes	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	130	Normal	M	4.38	38
201	Patient_201	21	7/19/2024	7/21/2024	Yes	72	12/1/2024	9/7/2025	G1P0	15	13739	64	31	2	2	1	5	0	5	No	Yes	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Negative	123	Normal	M	3.7	31
202	Patient_202	21	7/20/2024	7/23/2024	No	0	12/1/2024	9/7/2025	G1P0	24	12761	65	31	6	2	1	3	1	7	No	Yes	None	Yes	No	No	No		Induced with Oxytocin		Negative	99	TTN	M	3.74	36
203	Patient_203	31	7/21/2024	7/23/2024	Yes	68	12/1/2024	9/7/2025	G3P2	16	14514	66	24	5	1	0	14	1	7	No	Yes	None	Yes	No	No	No		Cesarean Section	Breech	Negative	134	TTN	M	3.97	38
204	Patient_204	19	7/22/2024	7/30/2024	No	0	12/1/2024	9/7/2025	G3P2	22	14209	52	24	8	1	1	1	4	7	No	No	Septicemia	No	No	No	No		Cesarean Section	Breech	Negative	62	RDS	F	3.67	33
205	Patient_205	23	7/23/2024	7/25/2024	No	0	12/1/2024	9/7/2025	G4P3	21	12975	59	21	7	2	1	4	7	8	No	Yes	Umbilical Sepsis	No	No	No	No		Cesarean Section	Fetal Distress	Negative	91	TTN	M	3.44	35
206	Patient_206	24	7/24/2024	1/8/2024	Yes	43	12/1/2024	9/7/2025	G3P2	20	29714	63	24	7	1	0	19	3	10	No	No	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	44	RDS	M	3.57	31
207	Patient_207	35	7/25/2024	7/27/2024	Yes	63	12/1/2024	9/7/2025	G3P2	18	13362	65	25	6	2	0	11	9	8	No	Yes	Conjunctivitis	No	No	No	No		Spontaneous		Negative	117	TTN	F	2.95	39
208	Patient_208	35	7/26/2024	7/29/2024	Yes	40	12/1/2024	9/7/2025	G4P3	14	11876	68	28	5	2	1	12.1	8	8	No	Yes	None	No	No	No	Yes	Prematurity	Cesarean Section	Placenta Previa	Negative	133	NORMAL	M	3.65	37
209	Patient_209	27	7/27/2024	8/9/2024	Yes	58	12/1/2024	9/7/2025	G1P0	18	14812	69	30	2	1	0	13.7	4	6	No	No	Septicemia	Yes	No	No	No		Induced with Oxytocin		Klebsiella	68	Normal	M	3.3	36
210	Patient_210	35	7/28/2024	7/31/2024	No	0	12/1/2024	9/7/2025	G3P2	23	14448	69	31	2	2	1	5	8	8	No	Yes	Conjunctivitis	No	No	No	No		Cesarean Section	Fetal Distress	Negative	138	Normal	F	3.07	33
211	Patient_211	35	7/29/2024	7/31/2024	No	0	12/1/2024	9/7/2025	G3P2	23	12312	61	34	6	2	0	11	9	8	No	Yes	None	No	No	No	No		Spontaneous		Negative	64	Normal	F	3.33	40
212	Patient_212	35	7/30/2024	8/2/2024	Yes	41	12/1/2024	9/7/2025	G1P0	21	12460	58	30	3	1	0	17	8	8	No	Yes	Conjunctivitis	No	Yes	No	No		Cesarean Section	Breech	Negative	136	HMD	F	2.09	28
213	Patient_213	24	7/31/2024	8/14/2024	Yes	49	12/1/2024	9/7/2025	G1P0	22	13709	58	37	3	2	1	14.7	4	7	No	No	Umbilical Sepsis	Yes	No	No	No		Induced with Oxytocin		Klebsiella	128	Normal	F	2.95	33
214	Patient_214	35	1/8/2024	1/11/2024	Yes	89	12/1/2024	9/7/2025	G4P3	21	13324	65	39	3	1	1	9.7	0	7	No	Yes	Umbilical Sepsis	Yes	No	No	No		Spontaneous		Negative	92	TTN	M	3.67	39
215	Patient_215	35	2/8/2024	2/19/2024	Yes	47	12/1/2024	9/7/2025	G1P0	20	12733	66	20	6	1	0	14	1	7	No	No	None	Yes	No	No	No		Induced with Oxytocin		Streptococcus	98	HMD	M	4.22	28
216	Patient_216	35	3/8/2024	3/11/2024	Yes	46	12/1/2024	9/7/2025	G1P0	16	12605	68	23	6	1	0	12	4	6	No	Yes	None	Yes	No	No	No		Spontaneous		Negative	53	RDS	F	2.46	31
217	Patient_217	32	4/8/2024	4/10/2024	No	0	12/1/2024	9/7/2025	G2P1	20	12526	55	37	4	1	1	2	9	8	No	Yes	Conjunctivitis	No	No	No	No		Cesarean Section	Placenta Previa	Negative	129	RDS	F	3.14	30
218	Patient_218	35	5/8/2024	12/8/2024	Yes	82	12/1/2024	9/7/2025	G1P0	14	23645	51	35	3	1	0	7.8	6	10	No	No	Pneumonia	No	No	No	No		Cesarean Section	Breech	Negative	93	NORMAL	F	2.83	36
219	Patient_219	35	6/8/2024	7/8/2024	No	0	12/1/2024	9/7/2025	G3P2	15	14842	63	26	8	2	0	10.6	2	5	No	No	Umbilical Sepsis	No	No	No	No		Cesarean Section	Fetal Distress	Negative	135	Normal	F	3.89	36
220	Patient_220	24	7/8/2024	8/15/2024	No	0	12/1/2024	9/7/2025	G3

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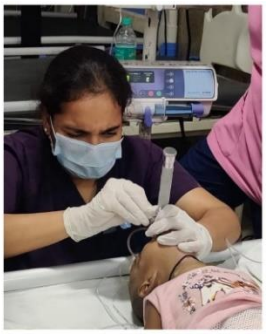
257	Patient_257	35	9/13/2024	9/14/2024	Yes	29	12/1/2024	9/7/2025	G2P1	16	16016	54	27	6	2	0	18	1	9	No	No	Umbilical Sepsis	No	No	No	No		Spontaneous		Negative	91	MAS	F	2.77	40
258	Patient_258	35	9/14/2024	9/17/2024	Yes	34	12/1/2024	9/7/2025	G4P3	14	13762	69	25	6	2	0	17	4	5	No	Yes	Umbilical Sepsis	Yes	No	No	No		Induced with Oxytocin		Negative	126	Normal	M	2.57	37
259	Patient_259	32	9/15/2024	9/27/2024	No	0	12/1/2024	9/7/2025	G3P2	22	12141	63	27	8	2	1	3	3	7	No	No	Umbilical Sepsis	Yes	No	No	No		Induced with Oxytocin		E. coli	91	NORMAL	M	3.21	37
260	Patient_260	28	9/16/2024	9/19/2024	Yes	29	12/1/2024	9/7/2025	G2P1	15	13883	52	27	6	2	1	2	7	8	No	Yes	Umbilical Sepsis	No	No	No	No		Cesarean Section	Placenta Previa	Negative	66	Normal	F	4.28	36
261	Patient_261	22	9/17/2024	9/29/2024	No	0	12/1/2024	9/7/2025	G2P1	24	12810	50	23	8	1	1	11	1	6	No	No	Conjunctivitis	Yes	No	No	No		Spontaneous		Streptococcus	70	Normal	M	2.26	32
262	Patient_262	27	9/18/2024	9/30/2024	No	0	12/1/2024	9/7/2025	G2P1	23	11723	64	20	2	1	0	3	0	7	No	No	None	Yes	No	No	No		Spontaneous		Streptococcus	42	RDS	M	3.43	30
263	Patient_263	35	9/19/2024	9/30/2024	Yes	29	12/1/2024	9/7/2025	G3P2	14	11224	52	39	6	2	0	3	3	5	No	No	Septicemia	Yes	No	No	No		Cesarean Section	Placenta Previa	Acinetobacter	106	RDS	F	4.25	31
264	Patient_264	20	9/20/2024	9/23/2024	Yes	72	12/1/2024	9/7/2025	G2P1	16	11531	52	34	6	2	0	5	7	8	No	Yes	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	121	TTN	M	2.49	39
265	Patient_265	35	9/21/2024	10/1/2024	No	0	12/1/2024	9/7/2025	G1P0	15	15208	70	31	7	2	0	20	5	5	No	No	Septicemia	Yes	No	No	No		Induced with Oxytocin		Negative	44	Normal	M	3.27	36
266	Patient_266	35	9/22/2024	9/25/2024	No	0	12/1/2024	9/7/2025	G3P2	18	13187	61	31	2	1	0	2	9	8	No	Yes	None	No	No	No	No		Spontaneous		Negative	40	NORMAL	M	2.13	38
267	Patient_267	18	9/23/2024	10/5/2024	No	0	12/1/2024	9/7/2025	G1P0	18	15654	57	35	4	2	0	14	4	6	No	No	None	Yes	No	No	No		Spontaneous		Acinetobacter	94	TTN	F	4.24	41
268	Patient_268	27	9/24/2024	9/26/2024	No	0	12/1/2024	9/7/2025	G3P2	22	29794	70	33	4	2	0	1	7	7	No	No	Pneumonia	No	No	No	No		Spontaneous		Negative	136	RDS	M	2.62	30
269	Patient_269	27	9/25/2024	10/6/2024	Yes	66	12/1/2024	9/7/2025	G4P3	24	13048	70	35	3	1	0	17	5	6	No	No	Conjunctivitis	Yes	No	No	No		Spontaneous		Streptococcus	93	RDS	M	3	33
270	Patient_270	32	9/26/2024	10/10/2024	No	0	12/1/2024	9/7/2025	G4P3	14	15749	62	36	4	2	1	10	3	6	No	No	Septicemia	Yes	No	No	No		Cesarean Section	Fetal Distress	Streptococcus	70	TTN	M	2.79	38
271	Patient_271	30	9/27/2024	10/7/2024	No	0	12/1/2024	9/7/2025	G1P0	21	15415	57	22	2	2	1	16	5	5	No	No	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	85	Normal	M	4.45	39
272	Patient_272	19	9/28/2024	10/9/2024	Yes	88	12/1/2024	9/7/2025	G3P2	21	12573	69	29	4	2	1	7.3	0	5	No	No	Conjunctivitis	Yes	No	No	No		Spontaneous		Klebsiella	119	RDS	F	3.05	33
273	Patient_273	28	9/29/2024	10/13/2024	No	0	12/1/2024	9/7/2025	G3P2	21	12828	62	40	5	1	0	12	4	6	No	No	None	Yes	No	No	No		Induced with Oxytocin		Streptococcus	47	Normal	M	2.78	34
274	Patient_274	35	9/30/2024	10/3/2024	Yes	29	12/1/2024	9/7/2025	G2P1	15	12917	68	30	4	2	0	12	9	8	No	Yes	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	135	NORMAL	F	3.23	39
275	Patient_275	35	1/10/2024	1/12/2024	No	0	12/1/2024	9/7/2025	G2P1	23	11138	61	37	8	2	0	16	7	8	No	Yes	None	No	No	No	No		Cesarean Section	Breech	Negative	108	RDS	M	3.72	30
276	Patient_276	23	2/10/2024	2/13/2024	No	0	12/1/2024	9/7/2025	G1P0	20	14582	51	30	4	1	0	2	9	8	No	Yes	Pneumonia	No	No	No	No		Cesarean Section	Fetal Distress	Negative	45	RDS	M	2.15	31
277	Patient_277	21	3/10/2024	4/10/2024	Yes	51	12/1/2024	9/7/2025	G1P0	18	17683	64	28	8	2	1	12.7	0	7	No	No	Umbilical Sepsis	No	No	No	No		Spontaneous		Negative	112	HMD	M	3.16	28
278	Patient_278	32	4/10/2024	4/13/2024	No	0	12/1/2024	9/7/2025	G1P0	23	13026	50	35	7	2	1	3	0	5	No	Yes	Conjunctivitis	Yes	No	No	No		Cesarean Section	Placenta Previa	Negative	92	RDS	F	3.73	30
279	Patient_279	35	5/10/2024	5/12/2024	Yes	61	12/1/2024	9/7/2025	G4P3	16	14744	51	25	8	2	0	17	3	7	No	Yes	None	Yes	No	No	No		Cesarean Section	Fetal Distress	Negative	121	MAS	M	4.45	40
280	Patient_280	35	6/10/2024	6/13/2024	Yes	89	12/1/2024	9/7/2025	G1P0	23	11901	67	24	7	2	0	9.7	1	5	No	Yes	Septicemia	Yes	No	No	No		Spontaneous		Negative	65	MAS	M	3.42	40
281	Patient_281	29	7/10/2024	10/14/2024	Yes	21	12/1/2024	9/7/2025	G1P0	14	18504	61	31	5	1	1	11.7	1	6	No	No	Septicemia	No	No	No	No		Spontaneous		Negative	116	MAS	M	3.28	39
282	Patient_282	30	8/10/2024	9/10/2024	No	0	12/1/2024	9/7/2025	G4P3	15	9699	64	31	2	2	0	11.7	3	8	No	No	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	43	TTN	M	2.6	38
283	Patient_283	35	9/10/2024	9/22/2024	Yes	23	12/1/2024	9/7/2025	G4P3	18	15785	68	32	8	2	0	3	4	5	No	No	Conjunctivitis	Yes	No	No	No		Induced with Oxytocin		E. coli	42	Normal	F	4.04	36
284	Patient_284	20	10/10/2024	10/13/2024	No	0	12/1/2024	9/7/2025	G3P2	24	13039	65	24	6	2	1	12	2	6	No	Yes	Conjunctivitis	Yes	No	No	No		Induced with Oxytocin		Negative	113	HMD	M	3.2	29
285	Patient_285	27	11/10/2024	11/12/2024	No	0	12/1/2024	9/7/2025	G4P3	21	11009	68	36	4	1	0	15	5	7	No	Yes	Umbilical Sepsis	Yes	No	No	No		Induced with Oxytocin		Negative	101	RDS	M	3.3	30
286	Patient_286	30	12/10/2024	12/20/2024	No	0	12/1/2024	9/7/2025	G1P0	19	11260	57	28	8	2	1	15	5	5	No	No	Conjunctivitis	Yes	No	No	No		Spontaneous		Negative	76	Normal	F	2.84	38
287	Patient_287	24	10/13/2024	10/18/2024	No	0	12/1/2024	9/7/2025	G1P0	16	13865	57	38	5	1	1	11	4	10	No	No	Conjunctivitis	No	No	No	No		Spontaneous		Negative	83	TTN	F	2.56	41
288	Patient_288	19	10/14/2024	10/23/2024	No	0	12/1/2024	9/7/2025	G4P3	24	28343	53	33	4	2	1	14	2	7	No	No	Umbilical Sepsis	No	No	No	No		Spontaneous		Negative	138	TTN	M	4.39	41
289	Patient_289	23	10/15/2024	10/22/2024	Yes	32	12/1/2024	9/7/2025	G3P2	14	28654	58	29	2	1	0	11	7	5	No	No	Pneumonia	No	No	No	No		Spontaneous		Negative	72	TTN	M	4.25	36
290	Patient_290	35	10/16/2024	10/19/2024	Yes	76	12/1/2024	9/7/2025	G1P0	24	12673	61	37	6	2	0	9	1	5	No	Yes	None	Yes	No	No	No		Spontaneous		Negative	135	RDS	M	4.14	32
291	Patient_291	32	10/17/2024	10/29/2024	No	0	12/1/2024	9/7/2025	G4P3	20	13219	61	22	3	1	0	17	3	7	No	No	Umbilical Sepsis	Yes	No	No	No		Induced with Oxytocin		Acinetobacter	118	Normal	F	3.52	35
292	Patient_292	35	10/18/2024	10/21/2024	Yes	75	12/1/2024	9/7/2025	G1P0	14	13945	57	22	4	2	0	7.8	7	8	No	Yes	Pneumonia	No	No	No	No		Cesarean Section	Breech	Negative	49	MAS	F	3.57	40
293	Patient_293	31	10/19/2024	10/30/2024	Yes	19	12/1/2024	9/7/2025	G4P3	16	13060	64	38	6	2	1	5	5	5	No	No	Conjunctivitis	Yes	No	No	No		Induced with Oxytocin		Streptococcus	126	NORMAL	F	4.2	34
294	Patient_294	22	10/20/2024	11/1/2024	No	0	12/1/2024	9/7/2025	G1P0	21	14713	57	30	8	2	1	18	2	5	No	No	Conjunctivitis	Yes	No	No	No		Spontaneous		Streptococcus	80	NORMAL	F	3.36	39
295	Patient_295	35	10/21/2024	10/31/2024	Yes	55	12/1/2024	9/7/2025	G3P2	16	12619	51	36	7	1	1	1	5	6	No	No	Conjunctivitis	Yes	No	No	No		Cesarean Section	Fetal Distress	Negative	81	RDS	F	2.18	30
296	Patient_296	27	10/22/2024	10/23/2024	No	0	12/1/2024	9/7/2025	G4P3	16	11327	52	20	4	1	0	4	5	6	No	No	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	95	NORMAL	F	2.88	37
297	Patient_297	24	10/23/2024	11/6/2024	No	0	12/1/2024	9/7/2025	G2P1	20	11320	59	21	5	1	1	11.1	2	6	No	No	Pneumonia	Yes	No	No	No		Spontaneous		E. coli	106	Abnormal	M	4.25	35
298	Patient_298	21	10/24/2024	10/27/2024	Yes	75	12/1/2024	9/7/2025	G1P0	17	12342	53	26	7	2	1	7.8	0	6	No	Yes	Conjunctivitis	Yes	No	No	No		Induced with Oxytocin		Negative	85	HMD	F	2.86	30
299	Patient_299	35	10/25/2024	10/27/2024	No	0	12/1/2024	9/7/2025	G2P1	23	12636	52	26	6	1	1	12	7	8	No	Yes	Conjunctivitis	No	No	No	No		Spontaneous		Negative	135	TTN	M	3.17	35
300	Patient_300	24	10/26/2024	11/7/2024	Yes	29	12/1/2024	9/7/2025	G4P3	23	13376	58	34	2	1	1	5	0	6	No	No	Septicemia	Yes	No	No	No		Induced with Oxytocin		Klebsiella	81	RDS	M	3.24	30
301	Patient_301	35	10/27/2024	11/6/2024	Yes	61	12/1/2024	9/7/2025	G4P3	18	13832	50	33	6	2	0	18	2	6	No	No	Umbilical Sepsis	Yes	No	No	No		Spontaneous		Negative	83	Normal	F	4.1	40
302	Patient_302	35	10/28/2024	10/31/2024	No	0	12/1/2024	9/7/2025	G1P0	18	13824	62	40	2	2	0	1	7	8	No	Yes	Umbilical Sepsis	No	No	No	No		Spontaneous		Negative	58	RDS	M	3.45	30
303	Patient_303	23	10/29/2024	10/31/2024	No	0	12/1/2024	9/7/2025	G3P2	23	12994	54	32	6	1	0	15	4	6	No	Yes	Septicemia	Yes	No	No	No		Induced with Oxytocin		Negative	63	Normal	F	2.08	41
304	Patient_304	22	10/30/2024	4/11/2024	Yes	76	12/1/2024	9/7/2025	G4P3	15	23415	58	20	5	1	0	6.6	6	7	No	No	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	82	RDS	F	2.72	32
305	Patient_305	19	10/31/2024	11/13/2024	Yes	72	12/1/2024	9/7/2025	G2P1	18	13958	61	30	2	1	1	10.9	0	5	No	No	Septicemia	Yes	No	No	No		Induced with Oxytocin		Klebsiella	127	Normal	M	4.19	37
306	Patient_306	19	1/11/2024	1/14/2024	Yes	32	12/1/2024	9/7/2025	G2P1	16	12943	52	32	3	2	1	5	9	8	No	Yes	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	43	NORMAL	F	3.39	39
307	Patient_307	31	2/11/2024	11/11/2024	No	0	12/1/2024	9/7/2025	G3P2	14	13216	64	26	7	1	0	12.5	8	6	No	No	None	No	Yes	No	No		Induced with Oxytocin		Negative	84	HMD	M	3.2	28
308	Patient_308	29	3/11/2024	3/14/2024	Yes	33	12/1/2024	9/7/2025	G4P3	20	14621	57	29	8	1	0	18	8	8	No	Yes	Pneumonia	No	No	No	No		Spontaneous		Negative	88	HMD	F	3.96	28
309	Patient_309	28	4/11/2024	11/13/2024	No	0	12/1/2024	9/7/2025	G1P0	18	16423	51	36	2	1	0	12.1	8	7	No	No	Septicemia	No	No	No	No		Spontaneous		Negative	58	Normal	F	3.55	41
310	Patient_310	22	5/11/2024	7/11/2024	Yes	89	12/1/2024	9/7/2025	G4P3	16	16366	66	25	4	1	1	9.7	0	6	No	No	None	No	No	No	No		Spontaneous		Negative	106	Normal	F	3.84	39
311	Patient_311	24	6/11/2024	6/13/2024	Yes	62	12/1/2024	9/7/2025	G1P0	23	13038	62	30	2	2	1	18	7	8	No	Yes	Septicemia	No	No	No	No		Cesarean Section	Breech	Negative	86	TTN	F	2.37	36
312	Patient_312	34	7/11/2024	7/23/2024	Yes	48	12/1/2024	9/7/2025	G3P2	20	14490	51	37	2	1	0	3	4	7	No	No	Umbilical Sepsis	Yes	No	No	No		Induced with Oxytocin		Klebsiella	93	Normal	F	2.98	32
313	Patient_313	28	8/11/2024	8/23/2024	Yes	48	12/1/2024	9/7/2025	G2P1	21	14677	58	38	4	2	0	4	1	7	No	No	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Klebsiella	136	Normal	F	2.23	37
314	Patient_314	35	9/11/2024	11/13/2024	Yes	89	12/1/2024	9/7/2025	G2P1	24	28498	68	30	7	2	0	7.4	6	10	No	No	Conjunctivitis	No	No	No	No		Spontaneous		Negative	99	TTN	F	3.35	35
315	Patient_315	34	10/11/2024	10/13/2024	No	0	12/1/2024	9/7/2025	G3P2	14	12118	68	34	7	2	1	4	1	6	No	Yes	Pneumonia	Yes	No	No	No		Induced with Oxytocin		Negative	132	NORMAL	F	3.89	39
316	Patient_316	31	11/11/2024	11/14/2024	Yes	37	12/1/2024	9/7/2025	G2P1	16	12604	58	21	6	2	0	15	5	5	No	Yes	None	Yes	No	No	No		Induced with Oxytocin		Negative	108	NORMAL	M	3.64	36
317	Patient_317	35	12/11/2024	12/13/2024	No	0	12/1/2024	9/7/2025	G4P3	16	13365	52	33	8	2	0	4	9	8	No	Yes	Pneumonia	No	No	No	No		Cesarean Section	Fetal Distress	Negative	131	RDS	F	2.76	32
318	Patient_318	24	11/13/2024	11/19/2024	Yes	37	12/1/2024	9/7/2025	G4P3	18	19152	53	40	7	1	0	12	6	8	No	No	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	86	NORMAL	M	2.93	40
319	Patient_319	35	11/14/2024	11/17/2024	Yes	64	12/1/2024	9/7/2025	G3P2	14	29098	70	25	2	2	0	11	8	10	No	No	Septicemia	No	No	No	No		Induced with Oxytocin		Negative	74	RDS	F	2.11	30
320	Patient_320	35	11/15/2024	11/26/2024	No	0	12/1/2024	9/7/2025	G4P3	18	15027	57	21	8	1	0	4	2	6	No	No	None	Yes	No	No	No		Spontaneous		E. coli	117	Normal	M	2.5	40
321	Patient_321	35	11/16/2024	11/18/2024	No	0	12/1/2024	9/7/2025	G2P1	19	11235	61	40	3	2	0	5	4	5	No	Yes	Umbilical Sepsis	Yes	No	No	No		Cesarean Section	Fetal Distress	Negative	118	NORMAL	F	2.47	38
322	Patient_322	30	11/17/2024	11/20/2024	No	0	12/1/2024	9/7/2025	G1P0	15	12969	51	36	4	1	0	17	0	5	No	Yes	Umbilical Sepsis	Yes	No	No	No		Spontaneous		Negative	45	NORMAL	M	4.21	31
323	Patient_323	22	11/18/2024	11/20/2024	No	0	12/1/2024	9/7/2025	G3P2	14	11333	60	31	8	1	0	1	8	8	No	Yes	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	137	HMD WITH IVH 1	M	3.53	29
324	Patient_324	31	11/19/2024	12/3/2024	Yes	31	12/1/2024	9/7/2025	G4P3	15	12908	61	23	2	2	0	12.9	4	6	No	No	Pneumonia	Yes	No	No	No		Spontaneous		E. coli	97	RDS	M	3.68	31
325	Patient_325	28	11/20/2024	11/21/2024	No	0	12/1/2024	9/7/2025	G3P2	18	19904	58	39	2	2	1	5	7	6	No	No	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	72	NORMAL	F	4.32	39
326	Patient_326	35	11/21/2024	11/23/2024	No	0	12/1/2024	9/7/2025	G4P3	22	10336	67	40	5	2	0	16	1	9	No	No	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	108	Normal	M	2.51	41
327	Patient_327	22	11/22/2024	11/24/2024	Yes	75	12/1/2024	9/7/2025	G3P2	16	15375	70	23	4	2	1	8.5	9	8	Yes	Yes	Pneumonia	No	No	Yes	No		Spontaneous		Negative	73	RDS	F	4.38	33
328	Patient_328	25	11/23/2024	11/26/2024	Yes	83	12/1/2024	9/7/2025	G2P1	23	14530	52	37	4	2	0	6.1	7	8	No	Yes	None	No	No	No	No		Cesarean Section	Placenta Previa	Negative	42	HMD WITH IVH 2	F	4.23	29
329	Patient_329	31	11/24/2024	3/12/2024	No	0	12/1/2024	9/7/2025	G4P3	15	20387	61	35	6	2	0	15	1	5	No	No	Septicemia	No	No	No	No		Induced with Oxytocin		Negative	47	RDS	M	2.34	30
330	Patient_330	35	11/25/2024	11/28/2024	No	0	12/1/2024	9/7/2025	G3P2	24	12160	57	21	7	2	1	12	9	8	No	Yes	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	93	HMD WITH IVH 1	M	2.41	28
331	Patient_331	35	11/26/2024	5/12/2024	Yes	81	12/1/2024	9/7/2025	G4P3	16	11844	67	26	8	1	1	8.7	5	7	No	No	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	123	Normal	M	2.47	38
332	Patient_332	30	11/27/2024	12/8/2024	Yes	31	12/1/2024	9/7/2025	G3P2	16	13635	63	28	5	1	1	2	2	5	No	No	Septicemia	Yes	No	No	No		Spontaneous		Acinetobacter	73	RDS	M	3.61	32
333	Patient_333	29	11/28/2024	12/12/2024	Yes	70	12/1/2024	9/7/2025	G3P2	24	12570	61	24	5	2	1	10.9	0	7	No	No	Septicemia	Yes	No	No	No		Cesarean Section	Fetal Distress	Klebsiella	64	Normal	M	4.17	38
334	Patient_334	24	11/29/2024	12/2/2024	No	0	12/1/2024	9/7/2025	G3P2	24	13560	70	27	4	1	1	16	7	8	No	Yes	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	64	NORMAL	M	2.92	37
335	Patient_335	21	11/30/2024	12/2/2024	No	0	12/1/2024	9/7/2025	G1P0	16	12319	60	23	8	2	1	1	8	8	No	Yes	None	No	No	No	No		Induced with Oxytocin		Negative	130	NORMAL	F	4.32	41
336	Patient_336	26	1/12/2024	4/12/2024	Yes	26	12/1/2024	9/7/2025	G4P3	24	26037	56	30	2	2	1	14.8	5	9	No	No	Septicemia	No	No	No	No		Cesarean Section	Breech	Negative	127	HMD	F	2.83	28
337	Patient_337	30	2/12/2024	5/12/2024	No	0	12/1/2024	9/7/2025	G4P3	24	28556	58	26	3	1	1	10.7	4	5	No	No	Umbilical Sepsis	No	No	No	No		Spontaneous		Negative	64	NORMAL	F	3.18	35
338	Patient_338	33	3/12/2024	11/12/2024	Yes	65	12/1/2024	9/7/2025	G4P3	20	17960	70	23	3	2	1	12.1	9	5	No	No	Septicemia	No	No	No	No		Spontaneous		Negative	57	Normal	M	3.56	34
339	Patient_339	35	4/12/2024	4/14/2024	Yes	65	12/1/2024	9/7/2025	G3P2	20	14391	50	26	7	2	0	15	8	8	No	Yes	Septicemia	No	No	No	No		Spontaneous		Negative	72	Normal	F	4.13	40
340	Patient_340	35	5/12/2024	5/15/2024	No	0	12/1/2024	9/7/2025	G2P1	20	13965	57	20	6	2	0	3	7	8	No	Yes	Pneumonia	No	No	No	No		Induced with Oxytocin		Negative	126	NORMAL	M	4.08	40
341	Patient_341	34	6/12/2024	12/12/2024	Yes	69	12/1/2024	9/7/2025	G2P1	24	28322	68	34	6	1	1	10.4	7	8	No	No	None	No	No	No	No		Cesarean Section	Fetal Distress	Negative	69	NORMAL	M	3.91	37
342	Patient_342	30	7/12/2024	7/15/2024	No	0	12/1/2024	9/7/2025	G1P0	16	14643	63	38	2	2	1	16	9	8	No	Yes	Septicemia	No	Yes	No	No		Spontaneous		Negative	114	HMD	F	4.44	28
343	Patient_343	22	8/12/2024	8/14/2024	No	0	12/1/2024	9/7/2025	G2P1	17	14850	56	38	7	1	1	15	0	5	No	Yes	Septicemia	Yes	No	No	No		Spontaneous		Negative	96	Normal	F	2.26	35
344	Patient_344	35	9/12/2024	9/15/2024	Yes	89	12/1/2024	9/7/2025	G1P0	19	11872	62	34	8	1	1	10	9	8	No	Yes	Umbilical Sepsis	No	No	No	No		Induced with Oxytocin		Negative	42	TTN	F	2.08	34
345	Patient_345	35	10/12/2024	10/14/2024	Yes	85	12/1/2024	9/7/2025	G4P3	21	12252	55	21	3	1	1	8	2	6	No	Yes	Septicemia	Yes	No	No	No		Induced with Oxytocin		Negative	70	Normal	M	3.94	35
346	Patient_346	23	11/12/2024	11/22/2024	No	0	12/1/2024	9/7/2025	G2P1	15	12314	70	38	2	2	1	12	3	7	No	No	Conjunctivitis	Yes	No	No	No		Induced with Oxytocin		Negative	45	Normal	M	2.83	33
347	Patient_347	29	12/12/2024	12/20/2024	Yes	89	12/1/2024	9/7/2025	G2P1	17	25697	66	24	5	1	0	8.4	2	8	No	No	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	71	TTN	M	2.09	36
348	Patient_348	35	12/13/2024	12/16/2024	No	0	12/1/2024	9/7/2025	G3P2	17	11724	58	29	7	1	1	14	8	8	No	Yes	Conjunctivitis	No	No	No	No		Induced with Oxytocin		Negative	105	Normal	M	2.65	37
349	Patient_349	35	12/14/2024	12/26/2024	No	0	12/1/2024	9/7/2025	G4P3	21	13898	69	39	7	1	1	4	2	5	No	No	None	Yes	No	No	No		Cesarean Section	Breech	Acinetobacter	74	Normal	F	3.36	33
350	Patient_350	35	12/15/2024	12/18/2024	Yes	64	12/1/2024	9/7/2025	G3P2	16	14450	65	34	4	1	1	3	8	8	No	Yes	Pneumonia	No	No	No	No		Spontaneous		Negative	48	NORMAL	F	3.26	35
351	Patient_351	32	12/16/2024	12/28/2024	No	0	12/1/2024	9/7/2025	G2P1	16	11077	54	24	5	2	0	5	1	6	No	No	Pneumonia	Yes	No	No	No		Cesarean Section	Fetal Distress	Klebsiella	115	Normal	F	2.56	33
352	Patient_352	35	12/17/2024	12/25/2024	No	0	12/1/2024	9/7/2025	G1P0	23	19846	64	33	5	2	0	20	7	10

"Residency Unplugged"

-First cry to first step

I thrived on night shifts, newborn cries, and life-changing moments. Sleep is scarce, but purpose is plenty—helping tiny humans and their families keeps me going.



It's a profound experience to witness the joy and relief on the faces of the families I help during those pivotal moments.

This journey as a pediatrician - in - training fills me with a sense of purpose and dedication to provide the best possible care.

Long hours, big impact, no regrets!

- Dr Shubhangi Bansal